

THE AMERICAN JOURNAL OF PSYCHIATRY

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In this issue:

Psychiatric Care and Health Insurance Reform

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The Phenomenological and Conceptual Interface
Between Borderline Personality Disorder and PTSD

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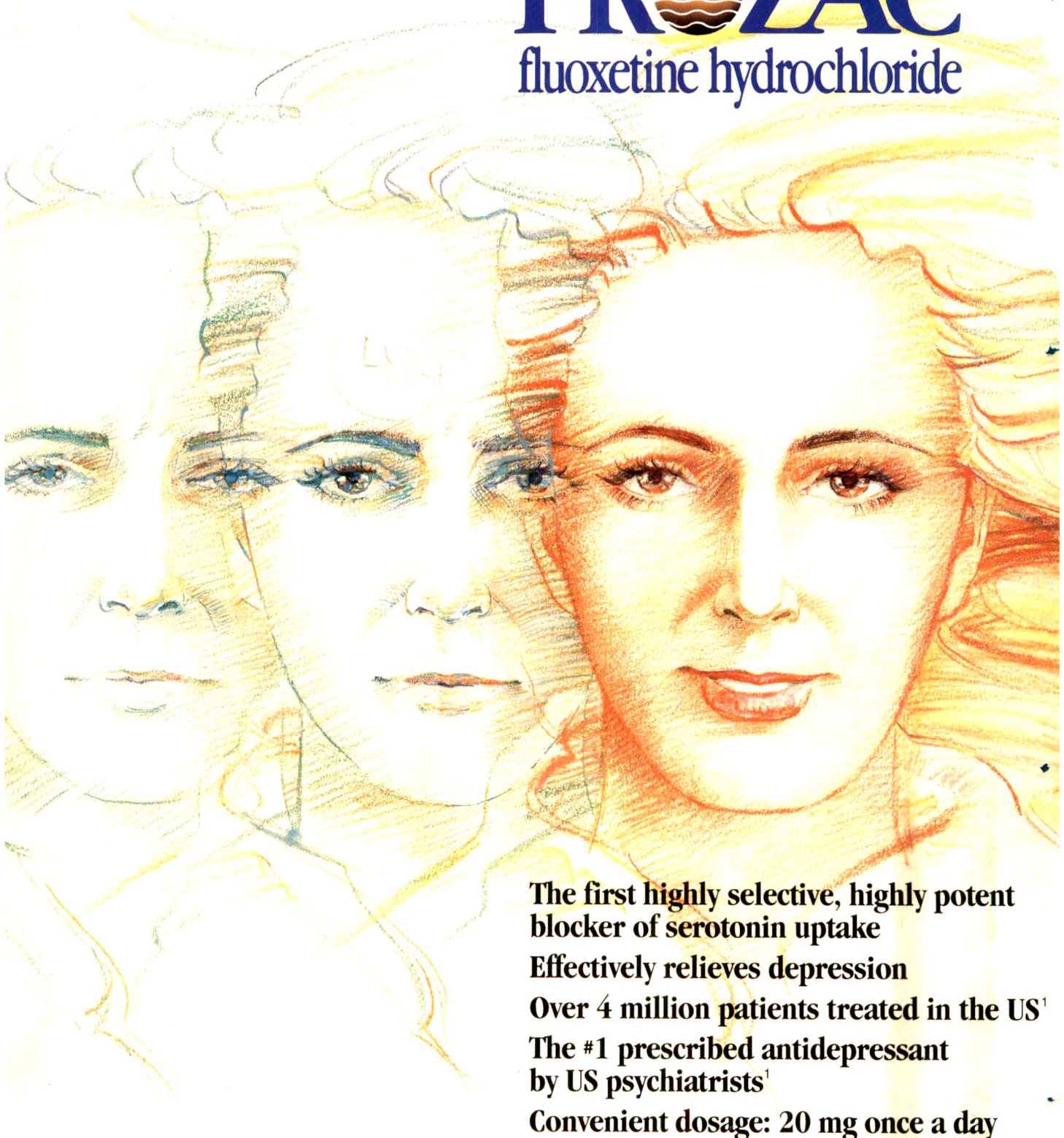
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By Marc Galanter

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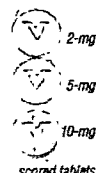
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BRIEF SUMMARY

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INDICATIONS AND USAGE: ZOLOFT (sertraline hydrochloride) is indicated for the treatment of depression.

CONTRAINDICATIONS: None known. **WARNINGS:** In patients receiving another serotonergic antidepressant, there have been reports of serious, sometimes fatal, reactions including hyperthermia, rigidity, myoclonus, autonomic instability with possible rapid fluctuations of vital signs, and mental status changes that include extreme agitation progressing to delirium and coma. These reactions have also been reported in patients who have recently discontinued that drug and have been started on an MAOI. Some cases presented with features resembling neuroleptic malignant syndrome. Therefore, it is recommended that ZOLOFT (sertraline hydrochloride) not be used in combination with an MAOI, or within 14 days of discontinuing treatment with an MAOI. Similarly, at least 14 days should be allowed after stopping ZOLOFT before starting an MAOI.

PRECAUTIONS General: Activation of Mania/Hypomania - During premarketing testing, hypomania or mania occurred in approximately 0.4% of ZOLOFT (sertraline hydrochloride) treated patients. Activation of mania/hypomania has also been reported in a small proportion of patients with Major Affective Disorder treated with other marketed antidepressants.

Weight Loss: Significant weight loss may be an undesirable result of treatment with sertraline for some patients, but on average, patients in controlled trials had minimal, 1 to 2 pound weight loss, versus smaller changes on placebo. Only rarely have sertraline patients been discontinued for weight loss. **Seizure:** ZOLOFT has not been evaluated in patients with a seizure disorder. These patients were excluded from clinical studies during the product's premarket testing. Accordingly, like other antidepressants, ZOLOFT should be introduced with care in epileptic patients. **Suicide:** The possibility of a suicide attempt is inherent in depression and may persist until significant remission occurs. Close supervision of high risk patients should accompany initial drug therapy. Prescriptions for ZOLOFT should be written for the smallest quantity of tablets consistent with good patient management, in order to reduce the risk of overdose. **Weak Uteric Acid Effect:** ZOLOFT is associated with a mean decrease in serum uric acid of approximately 7%. The clinical significance of this weak uric acid effect is unknown, and there have been no reports of acute renal failure with ZOLOFT. **Use in Patients with Concomitant Illness:** Clinical experience with ZOLOFT in patients with certain concomitant systemic illness is limited. Caution is advisable in using ZOLOFT in patients with diseases or conditions that could affect metabolism or hemodynamic responses. ZOLOFT has not been evaluated or used to any appreciable extent in patients with a recent history of myocardial infarction or unstable heart disease. Patients with these diagnoses were excluded from clinical studies during the product's premarket testing. However, the electrocardiograms of 774 patients who received ZOLOFT in double-blind trials were evaluated and the data indicate that ZOLOFT is not associated with the development of significant ECG abnormalities. ZOLOFT is extensively metabolized by the liver. The pharmacokinetics of ZOLOFT have not been studied in patients with significant hepatic dysfunction nor have patients with significant hepatic dysfunction been evaluated during treatment with ZOLOFT. Accordingly, ZOLOFT should be used with caution in such patients. Since ZOLOFT is extensively metabolized, excretion of unchanged drug in urine is minor route of elimination. However, until the pharmacokinetics of ZOLOFT have been studied in patients with renal impairment and until adequate numbers of patients with severe renal impairment have been evaluated during chronic treatment with ZOLOFT, it should be used with caution in such patients. **Interference with Cognitive and Motor Performance:** In controlled studies, ZOLOFT did not cause sedation and did not interfere with psychomotor performance. **Information for Patients:** Physicians are advised to discuss the following issues with patients for whom they prescribe ZOLOFT. Patients should be told that although ZOLOFT has not been shown to impair the ability of normal subjects to perform tasks requiring complex motor and mental skills in laboratory experiments, drugs that act upon the central nervous system may affect some individuals adversely. Patients should be told that although ZOLOFT has not been shown in experiments with normal subjects to increase the mental and motor skill impairments caused by alcohol, the concomitant use of ZOLOFT and alcohol in depressed patients is not advised. Patients should be told that while no adverse interaction of ZOLOFT with over-the-counter (OTC) drug products is known to occur, the potential for interaction exists. Thus, the use of any OTC product should be initiated cautiously according to the directions of use given for the OTC product. Patients should be advised to notify their physician if they become pregnant or intend to become pregnant during therapy. Patients should be advised to notify their physician if they are breast-feeding an infant. **Laboratory Tests:** None. **Drug Interactions: Potential Effects of Coadministration of Drugs Highly Bound to Plasma Proteins:** Because sertraline is tightly bound to plasma protein, the administration of ZOLOFT (sertraline hydrochloride) to a patient taking another drug which is tightly bound to protein (e.g., warfarin, digoxin) may cause a shift in plasma concentrations potentially resulting in an adverse effect. Conversely, adverse effects may result from displacement of protein-bound ZOLOFT by other tightly-bound drugs. In a study comparing prothrombin time AUC (0-120 hr) following dosing with warfarin (0.75 mg/kg) before and after 21 days of dosing with either ZOLOFT (50-200 mg/day) or placebo, there was a mean increase in prothrombin time of 8% relative to baseline for ZOLOFT compared to a 1% decrease for placebo (p<0.02). The normalization of prothrombin time for the ZOLOFT group was delayed compared to the placebo group. The clinical significance of this change is unknown. Accordingly, prothrombin time should be carefully monitored when ZOLOFT therapy is initiated or stopped. **CNS Active Drugs:** In a study comparing the disposition of intravenously administered diazepam before and after 21 days of dosing with either ZOLOFT (50-200 mg/day escalating dose) or placebo, there was a 32% decrease relative to baseline in diazepam clearance for the ZOLOFT group compared to a 19% decrease relative to baseline for the placebo group (p<0.03). There was a 23% increase in T_{max} for desmethyldiazepam in the ZOLOFT group compared to a 20% decrease in the placebo group (p<0.03). The clinical significance of these changes is unknown. In a placebo-controlled trial in normal volunteers, the administration of two doses of ZOLOFT did not significantly alter steady-state lithium levels or the renal clearance of lithium. Nonetheless, at this time, it is recommended that plasma lithium levels be monitored following initiation of ZOLOFT therapy with appropriate adjustments to the lithium dose. The risk of using ZOLOFT in combination with other CNS active drugs has not been systematically evaluated. Consequently, caution is advised if the concomitant administration of ZOLOFT and such drugs is required. **Hypoglycemic Drugs:** In a placebo-controlled trial in normal volunteers, administration of ZOLOFT for 22 days (including 200 mg/day for the final 13 days) caused a statistically significant 16% decrease from baseline in the clearance of tolbutamide following an intravenous 1000 mg dose. ZOLOFT administration did not noticeably change either the plasma protein binding or the apparent volume of distribution of tolbutamide, suggesting that the decreased clearance was due to a change in the metabolism of the drug. The clinical significance of this decrease in tolbutamide clearance is unknown. **Atenolol:** ZOLOFT (100 mg) when administered to 10 healthy male subjects had no effect on the beta-adrenergic blocking ability of atenolol. **Microssomal Enzyme Induction:** Preliminary studies have shown ZOLOFT to induce hepatic microssomal enzymes. In clinical studies ZOLOFT was shown to induce hepatic enzymes minimally as determined by a small (5%) but statistically significant decrease in antipyrine half-life following administration of 200 mg/day for 21 days. This small change in antipyrine half-life reflects a clinically insignificant change in hepatic metabolism. **Electroconvulsive Therapy:** There are no clinical studies establishing the risks or benefits of the combined use of electroconvulsive therapy (ECT) and ZOLOFT. **Alcohol:** Although ZOLOFT did not potentiate the cognitive and psychomotor effects of alcohol in experiments with normal subjects, the concomitant use of ZOLOFT and alcohol in depressed patients is not recommended. **Carcinogenesis, Mutagenesis, Impairment of Fertility:** Lifetime carcinogenicity studies were carried out in CD-1 mice and Long-Evans rats at doses up to 40 mg/kg in mice (10 times on a mg/kg basis and the same on a mg/m² basis as the maximum recommended human dose) and at doses up to 40 mg/kg in rats (10 times on a mg/kg basis and 2 times on a mg/m² basis; the maximum recommended human dose). There was a dose-related increase in the incidence of liver adenomas in male mice receiving sertraline at 10-40 mg/kg. No increase was seen in female mice or in rats of either sex receiving the same treatments, nor was there an increase in hepatocellular carcinomas. Liver adenomas have a variable rate of spontaneous occurrence in the 40-100 mg/kg range and are of unknown significance to humans. There was an increase in follicular adenomas of the thyroid in female rats receiving sertraline at 40 mg/kg; this was not accompanied by thyroid hyperplasia. While there was an increase in uterine adenocarcinomas in rats receiving sertraline at 10-40 mg/kg compared to placebo controls, this effect was not clearly drug related. Sertraline had no genotoxic effects, with or without metabolic activation, based on the following assays: bacterial mutation assay; mouse lymphoma mutation assay; and tests for cytogenetic aberrations *in vivo* in mouse bone marrow and *in vitro* in human lymphocytes. A decrease in fertility was seen in one of two rat studies at a dose of 80 mg/kg (20 times the maximum human dose) in a mg/kg basis and 4 times on a mg/m² basis). **Pregnancy—Pregnancy Category B:** Teratogenic Effects - Reproduction studies have been performed in rats and rabbits at doses up to approximately 20 times and 10 times the maximum dose (human mg/kg dose (4 to 4.5 times the mg/m² dose), respectively. There was no evidence of teratogenicity at any dose level. At doses approximately 2.5-10 times the maximum daily human mg/kg dose, sertraline was associated with delayed ossification in fetuses, probably secondary to effects on the dams. There are no adequate and well-controlled studies in pregnant women. Because animal reproduction studies are not always predictive of human response, this drug should be used during pregnancy only if clearly needed. **Non-teratogenic Effects:** There was also decreased neonatal survival following maternal administration of sertraline at doses as low as approximately 5 times the maximum human mg/kg dose. The decrease in pup survival was shown to be most probably due to *in utero* exposure to sertraline. The clinical significance of these effects is unknown. **Laboratory and Delivery:** The effect of ZOLOFT on labor and delivery in humans is unknown. **Nursing Mothers:** It

is not known whether, and if so in what amount, sertraline or its metabolites are excreted in human milk. Because many drugs are excreted in human milk, caution should be exercised when ZOLOFT is administered to a nursing woman. **Pediatric Use - Safety and effectiveness in children have not been established. Geriatric Use -** Several hundred elderly patients have participated in clinical studies with ZOLOFT. The pattern of adverse reactions in the elderly was similar to that in younger patients. **ADVERSE REACTIONS Commonly Observed:** The most commonly observed adverse events associated with the use of ZOLOFT (sertraline hydrochloride) and not seen at an equivalent incidence among placebo-treated patients were: gastrointestinal complaints, including nausea, diarrhea/loose stools and dyspepsia; tremor; dizziness; insomnia; somnolence; increased sweating; dry mouth; and male sexual dysfunction (primarily ejaculatory delay). **Associated with Discontinuation of Treatment:** Fifteen percent of 2710 subjects who received ZOLOFT in premarketing multiple dose clinical trials discontinued treatment due to an adverse event. The most common events (reported by at least 1% of subjects) associated with discontinuation included agitation, insomnia, male sexual dysfunction (primarily ejaculatory delay), somnolence, dizziness, headache, tremor, anorexia, diarrhea/loose stools, nausea, and fatigue. **Incidence in Controlled Clinical Trials:** The table that follows enumerates adverse events that occurred at a frequency of 1% or more among ZOLOFT patients who participated in controlled trials comparing titrated ZOLOFT with placebo. Most patients received doses of 50 to 200 mg per day.

Treatment-Emergent Adverse Experience Incidence in Placebo-Controlled Clinical Trials*							
Adverse Experience		(Percent of Patients Reporting)		Adverse Experience		(Percent of Patients Reporting)	
		ZOLOFT (N=861)	Placebo (N=853)			ZOLOFT (N=861)	Placebo (N=853)
Autonomic Nervous System Disorders							
Mouth Dry		16.3	9.3	Hot Flashes		2.2	0.5
Sweating Increased		8.4	2.9	Fever		1.6	0.6
				Back Pain		1.5	0.9
Cardiovascular							
Palpitations		3.5	1.6	Thirst		1.4	0.9
Chest Pain		1.0	1.6	Musculoskeletal System Disorders			
				Myalgia		1.7	1.5
Central and Peripheral Nervous System Disorders							
Disorders							
Headache		20.3	19.0	Insomnia		16.4	8.8
Dizziness		11.7	6.7	Sexual Dysfunction-Male (1)		15.5	2.2
Tremor		10.7	2.7	Somnolence		13.4	5.9
Paresthesia		2.0	1.8	Agitation		5.6	4.0
Hypoesthesia		1.7	0.6	Nervousness		3.4	1.9
Twitching		1.4	0.1	Anxiety		2.6	1.3
Hypertonia		1.3	0.4	Yawning		1.9	0.2
Disorders of Skin and Appendages							
Rash		2.1	1.5	Sexual Dysfunction-Female (2)		1.7	0.2
				Concentration Impaired		1.3	0.5
Gastrointestinal Disorders							
Nausea		26.1	11.8	Reproductive			
Diarrhea/Loose Stools		17.7	9.3	Menstrual Disorder (2)		1.0	0.5
Constipation		8.4	6.3	Respiratory System Disorders			
Dyspepsia		6.0	2.8	Rhinitis		2.0	1.5
Vomiting		3.8	1.8	Pharyngitis		1.2	0.9
Special Senses							
Flatulence		3.3	2.5	Vision Abnormal		4.2	2.1
Anorexia		2.8	1.6	Tinnitus		1.4	1.1
Abdominal Pain		2.4	2.2	Taste Perversion		1.2	0.7
Urinary System Disorders							
Appetite Increased		1.3	0.9	Micturition Frequency		2.0	1.2
				Micturition Disorder		1.4	0.5
General							
Fatigue		10.6	8.1				

*Events reported by at least 1% of patients treated with ZOLOFT are included.

(1) - % based on male patients only: 271 ZOLOFT (primarily ejaculatory delay) and 271 placebo patients.

(2) - % based on female patients only: 590 ZOLOFT and 582 placebo patients.

Other Events Observed During the Premarketing Evaluation of ZOLOFT (sertraline hydrochloride): During its premarketing assessment, multiple doses of ZOLOFT were administered to approximately 2700 subjects. Events are further categorized by body system and listed in order of decreasing frequency according to the following definitions: frequent adverse events are those occurring on one or more occasions in at least 1/100 patients (only those not already listed in the tabulated results from placebo-controlled trials appear in this listing); infrequent adverse events are those occurring in 1/100 to 1/1000 patients; rare events are those occurring in fewer than 1/1000 patients. Events of major clinical importance are also described in the PRECAUTIONS section. **Autonomic Nervous System Disorders—Infrequent:** flushing, mydriasis, increased saliva, cold clammy skin; **Rare:** pallor. **Cardiovascular—Infrequent:** postural dizziness, hypertension, hypotension, postural hypotension, edema, dependent edema, periorbital edema, peripheral edema, peripheral ischemia, syncope, tachycardia; **Rare:** precordial chest pain, substernal chest pain, aggravated hypertension, myocardial infarction, varicose veins. **Central and Peripheral Nervous System Disorders—Frequent:** confusion; **Infrequent:** ataxia, abnormal coordination, abnormal gait, hyperesthesia, hyperkinesia, hypokinesia, migraine, nystagmus, vertigo; **Rare:** local anesthesia, coma, convulsions, dyskinesia, dysphonia, hyporeflexia, hypotonia, ptosis. **Disorders of Skin and Appendages—Infrequent:** acne, alopecia, pruritus, erythematous rash, maculopapular rash, dry skin; **Rare:** bullous eruption, dermatitis, erythema multiforme, abnormal hair texture, periorthostic photosensitivity reaction, follicular rash, skin discoloration, abnormal skin odor, urticaria. **Endocrine Disorders—Rare:** exophthalmos, gynecostoma. **Gastrointestinal Disorders—Infrequent:** dysphagia, eructation; **Rare:** diverticulitis, fecal incontinence, gastritis, gastroenteritis, glossitis, gum hyperplasia, hemorrhoids, hiccup, melena, hemorrhagic peptic ulcer, proctitis, stomatitis, ulcerative stomatitis, tenesmus, tongue edema, tongue ulceration. **General—Frequent:** asthenia; **Infrequent:** malaise, generalized edema, rigors, weight decrease, weight increase; **Rare:** enlarged abdomen, hiccups, otitis media, aphthous stomatitis. **Hematopoietic and Lymphatic—Infrequent:** lymphadenopathy, purpura; **Rare:** anemia, anterior chamber eye hemorrhage. **Metabolic and Nutritional Disorders—Rare:** dehydration, hypercholesterolemia, hypoglycemia. **Musculoskeletal System Disorders—Infrequent:** arthralgia, arthrosis, arthralgia, dizziness, muscle cramps, muscle weakness; **Rare:** hemia. **Psychiatric Disorders—Infrequent:** abnormal dreams, aggressive reaction, amnesia, apathy, delusion, depersonalization, depression, aggravated depression, emotional lability, euphoria, hallucination, neuritis, paranoid reaction, suicide ideation and attempt, teeth-grinding, abnormal thinking; **Rare:** hysteria, somnambulism, withdrawal syndrome. **Reproductive—Infrequent:** dysmenorrhea (2), intermenstrual bleeding (2); **Rare:** amenorrhea (2), balanoposthitis (1), breast enlargement (2), female breast pain (2), leukorrhea (2), menorrhagia (2), atrophic vaginitis (2).

(1) - % based on male subjects only: 1005; (2) - % based on female subjects only: 1705.

Respiratory System Disorders—Infrequent: bronchospasm, coughing, dyspnea, epistaxis; **Rare:** bradypnea, hyperventilation, sinusitis, stridor. **Special Senses—Infrequent:** abnormal accommodation, conjunctivitis, diplopia, earache, eye pain, xerophthalmia; **Rare:** abnormal lacrimation, photophobia, visual field defect. **Urinary System Disorders—Infrequent:** dysuria, face edema, nocturia, polyuria, urinary incontinence; **Rare:** oliguria, renal pain, urinary retention. **Laboratory Tests:** In man, asymptomatic elevations in serum transaminases (SGOT [or AST] and SGPT [or ALT]) have been reported infrequently (approximately 0.8%) in association with ZOLOFT administration. These hepatic enzyme elevations usually occurred within the first 1 to 3 weeks of drug treatment and promptly diminished upon drug discontinuation. ZOLOFT therapy was associated with small mean increases in total cholesterol (approximately 3%) and triglycerides (approximately 5%), and a small mean decrease in serum uric acid (approximately 7%) of no apparent clinical importance. **DRUG ABUSE AND DEPENDENCE Controlled Substance Class -** ZOLOFT (sertraline hydrochloride) is not a controlled substance. **Physical and Psychological Dependence -** ZOLOFT has not been systematically studied, in animals or humans, for its potential for abuse, tolerance, or physical dependence. However, the premarketing clinical experience with ZOLOFT did not reveal any tendency for a withdrawal syndrome or any drug-seeking behavior. As with any new CNS active drug, physicians should carefully evaluate patients for history of drug abuse and follow such patients closely, observing them for signs of ZOLOFT misuse or abuse (e.g., development of tolerance, incrementation of dose, drug-seeking behavior). **OVERDOSE Human Experience -** There have been 3 cases of ZOLOFT (sertraline hydrochloride) overdose (approximately 750-2,100 mg). No specific therapy was required for any of the 3 patients, all of whom recovered completely. **Management of Overdoses -** Establish and maintain an airway, insure adequate oxygenation and ventilation. Activated charcoal, which may be used with sorbitol, may be as or more effective than emesis or lavage, and should be considered in treating overdose. Cardiac and vital signs monitoring is recommended along with general symptomatic and supportive measures. There are no specific antidotes for ZOLOFT. Due to the large volume of distribution of ZOLOFT, forced diuresis, dialysis, hemoperfusion, and exchange transfusion are unlikely to be of benefit. In managing overdose, consider the possibility of multiple drug involvement. The physician should consider contacting a poison control center on the treatment of any overdose.



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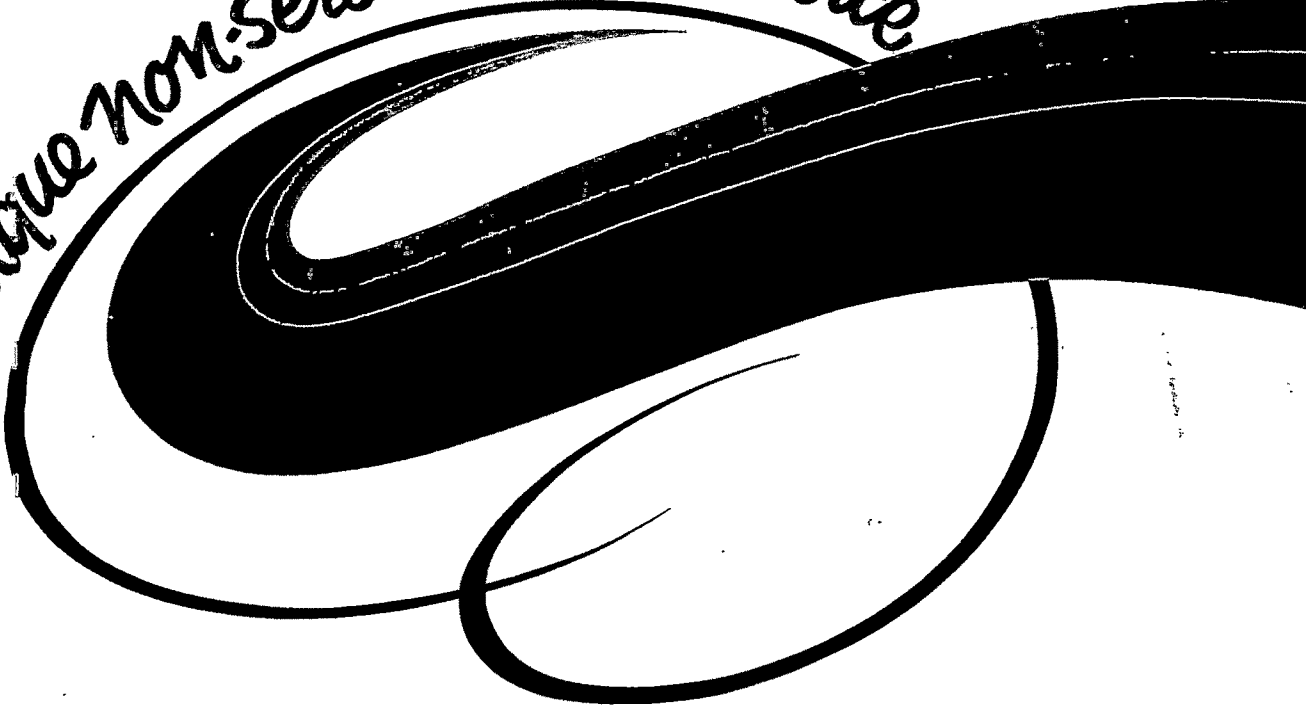
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* The principal medically important adverse reaction with WELLBUTRIN is seizure, which occurs in approximately four-tenths of one percent (4 out of 1000) of patients. This incidence may exceed that of other marketed antidepressants although no direct comparative studies have been conducted. For more information about dosing to achieve optimal patient response and recommendations for reducing the risk of side effects, see full prescribing information, especially the DOSAGE AND ADMINISTRATION and WARNINGS sections.

See brief summary of full prescribing information on last page of this advertisement.

† Prozac (fluoxetine HCl) is a registered trademark of Dista Products Co., a division of Eli Lilly and Company.

‡ Mean daily dose at week 6 was 382 mg for WELLBUTRIN patients and 38 mg for fluoxetine patients.

§ In a 3,341-patient surveillance study designed to determine the incidence of seizures with bupropion under conditions of general clinical practice, physicians were asked to report whether patients' responses to and tolerance of the antidepressant received in the month before the study were good, poor, or unknown. In the month before the study, 1,902 patients received previous antidepressant treatment. The results reported reflected evaluations of response to and tolerance of WELLBUTRIN at the end of up to 56 days of treatment.⁸

WELLBUTRIN® (BUPROPION HYDROCHLORIDE) Tablets

Before prescribing, please consult complete product information, a summary of which follows: **INDICATIONS AND USAGE:** Wellbutrin is indicated for the treatment of depression. A physician considering the initiation of Wellbutrin should be aware that the drug may cause generalized seizures with an approximate incidence of 0.4% (4/1000). This incidence may exceed that of other antidepressants as much as fourfold. This relative risk is only an approximation since no direct comparative studies have been conducted. **CONTRAINDICATIONS:** Wellbutrin is contraindicated in patients with a seizure disorder; with a current or prior diagnosis of bulimia or anorexia nervosa, because of a higher incidence of seizures noted in such patients; who have shown an allergic response to it; or who are currently being treated with an MAO inhibitor. At least 14 days should elapse between discontinuation of an MAO inhibitor and initiation of treatment with Wellbutrin.

WARNINGS: SEIZURES: Wellbutrin is associated with seizures in approximately 0.4% (4/1000) of patients treated at doses up to 450 mg/day. This incidence of seizures may exceed that of other marketed antidepressants by as much as fourfold. This relative risk is only an approximate estimate because no direct comparative studies have been conducted. The estimated seizure incidence for Wellbutrin increases almost tenfold between 450 and 600 mg/day, which is twice the usually required daily dose (300 mg) and one and one-third the maximum recommended daily dose (450 mg). Given the wide variability among individuals and their capacity to metabolize and eliminate drugs, this disproportionate increase in seizure incidence with dose incrementation calls for caution in dosing. During the initial development, 25 among approximately 2400 patients treated with Wellbutrin experienced seizures. At the time of seizure, 7 patients were receiving daily doses of 450 mg or below, for an incidence of 0.33% (3/1000) within the recommended dose range. Twelve (12) patients experienced seizures at 600 mg per day (2.3% incidence); 6 additional patients had seizures at daily doses between 600 and 900 mg (2.8% incidence).

A separate, prospective study was conducted to determine the incidence of seizure during an 8 week treatment exposure in approximately 3200 additional patients who received daily doses of up to 450 mg. Patients were permitted to continue treatment beyond 8 weeks if clinically indicated. Eight (8) seizures occurred during the initial 8 week treatment period and 5 seizures were reported in patients continuing treatment beyond 8 weeks, resulting in a total seizure incidence of 0.4%. The risk of seizure appears to be strongly associated with dose and the presence of predisposing factors. A significant predisposing factor (e.g., history of head trauma or prior seizure, CNS tumor, concomitant medications that lower seizure threshold, etc.) was present in approximately one-half of the patients experiencing a seizure. Sudden and large increments in dose may contribute to increased risk. While many seizures occurred early in the course of treatment, some seizures did occur after several weeks at fixed dose.

Recommendations for reducing the risk of seizure: Retrospective analysis of clinical experience gained during the development of Wellbutrin suggests that the risk of seizure may be minimized if (1) the total daily dose of Wellbutrin does not exceed 450 mg, (2) the daily dose is administered t.i.d., with each single dose not to exceed 150 mg to avoid high peak concentrations of bupropion and/or its metabolites, and (3) the rate of incrementation of dose is very gradual. Extreme caution should be used when Wellbutrin is (1) administered to patients with a history of seizure, cranial trauma, or other predisposition(s) toward seizure, or (2) prescribed with other agents (e.g., antipsychotics, other antidepressants, etc.) or treatment regimens (e.g., abrupt discontinuation of a benzodiazepine) that lower seizure threshold.

Potential for Hepatotoxicity: In rats receiving large doses of bupropion chronically, there was an increase in incidence of hepatic hyperplastic nodules and hepatocellular hypertrophy. In dogs receiving large doses of bupropion chronically, various histologic changes were seen in the liver, and laboratory tests suggesting mild hepatocellular injury were noted. Although scattered abnormalities in liver function tests were detected in patients participating in clinical trials, there is no clinical evidence that bupropion acts as a hepatotoxin in humans.

PRECAUTIONS: General:

Agitation and Insomnia: A substantial proportion of patients treated with Wellbutrin experience some degree of increased restlessness, agitation, anxiety, and insomnia, especially shortly after initiation of treatment. In clinical studies, these symptoms were sometimes of sufficient magnitude to require treatment with sedative/hypnotic drugs. In approximately 2% of patients, symptoms were sufficiently severe to require discontinuation of Wellbutrin treatment.

Psychosis, Confusion, and Other Neuropsychiatric Phenomena: Patients treated with Wellbutrin have been reported to show a variety of neuropsychiatric signs and symptoms including delusions, hallucinations, psychotic episodes, confusion, and paranoia. Because of the uncontrolled nature of many studies, it is impossible to provide a precise estimate of the extent of risk imposed by treatment with Wellbutrin. In several cases, neuropsychiatric phenomena abated upon dose reduction and/or withdrawal of treatment.

Activation of Psychosis and/or Mania: Antidepressants can precipitate manic episodes in Bipolar Manic Depressive patients during the depressed phase of their illness and may activate latent psychosis in other susceptible patients. Wellbutrin is expected to pose similar risks.

Altered Appetite and Weight: A weight loss of greater than 5 pounds occurred in 28% of Wellbutrin patients. This incidence is approximately double that seen in comparable patients treated with tricyclics or placebo. Furthermore, while 34.5% of patients receiving tricyclic antidepressants gained weight, only 9.4% of patients treated with Wellbutrin did. Consequently, if weight loss is a major presenting sign of a patient's depressive illness, the anorectic and/or weight reducing potential of Wellbutrin should be considered.

Suicide: The possibility of a suicide attempt is inherent in depression and may persist until significant remission occurs. Accordingly, prescriptions for Wellbutrin should be written for the smallest number of tablets consistent with good patient management.

Use in Patients with Systemic Illness: There is no clinical experience establishing the safety of Wellbutrin in patients with a recent history of myocardial infarction or unstable heart disease. Therefore, care should be exercised if it is used in these groups.

Because bupropion HCl and its metabolites are almost completely excreted through the kidney and metabolites are likely to undergo conjugation in the liver prior to urinary excretion, treatment of patients with renal or hepatic impairment should be initiated at reduced dosage as bupropion and its metabolites may accumulate in such patients beyond concentrations expected in patients without renal or hepatic impairment. The patient should be closely monitored for possible toxic effects of elevated blood and tissue levels of drug and metabolites.

Information for Patients: Consult complete product information.

Drug Interactions: No systematic data have been collected on the consequences of the concomitant administration of Wellbutrin and other drugs. However, animal data suggest that Wellbutrin may be an inducer of drug metabolizing enzymes. This may be of potential clinical importance because the blood levels of co-administered drugs may be altered. Alternatively, because bupropion is extensively metabolized, the co-administration of other drugs may affect its clinical activity. In particular, care should be exercised when administering drugs known to affect hepatic drug metabolizing enzyme systems (e.g., carbamazepine, cimetidine, phenobarbital, phenytoin). Studies in animals demonstrate that the acute toxicity of bupropion is enhanced by the MAO inhibitor phenelzine (see CONTRAINDICATIONS).

Limited clinical data suggest a higher incidence of adverse experiences in patients receiving concurrent administration of Wellbutrin and L-dopa. Administration of Wellbutrin to patients receiving L-dopa concurrently should be undertaken with caution, using small initial doses and small gradual dose increases. Concurrent administration of Wellbutrin and agents which lower seizure threshold should be undertaken only with extreme caution (see WARNINGS). Low initial dosing and small gradual dose increases should be employed.

Carcinogenesis, Mutagenesis, Impairment of Fertility: Lifetime carcinogenicity studies were performed in rats and mice at doses up to 300 and 150 mg/kg/day, respectively. In the rat study there was an increase in nodular proliferative lesions of the liver at doses of 100 to 300 mg/kg/day; lower doses were not tested. The question of whether or not such lesions may be precursors of neoplasms of the liver is currently unresolved. Similar liver lesions were not seen in the mouse study, and no increase in malignant tumors of the liver and other organs was seen in either study.

Bupropion produced a borderline positive response (2-3 times control mutation rate) in some strains in the Ames bacterial mutagenicity test, and a high oral dose (300, but not 100 or 200 mg/kg) produced a low incidence of chromosomal aberrations in rats. The relevance of these results in estimating the risk of human exposure to therapeutic doses is unknown.

A fertility study was performed in rats; no evidence of impairment of fertility was encountered at oral doses up to 300 mg/kg/day.

Pregnancy: Teratogenic Effects: Pregnancy Category B: Reproduction studies have been performed in rabbits and rats at doses up to 15-45 times the human daily dose and have revealed no definitive evidence of impaired fertility or harm to the fetus due to bupropion. (In rabbits, a slightly increased incidence of fetal abnormalities was seen in two studies, but there was no increase in any specific abnormality). There are no adequate and well-controlled studies in pregnant women. Because animal reproduction studies are not always predictive of human response, this drug should be used during pregnancy only if clearly needed.

Labor and Delivery: The effect of Wellbutrin on labor and delivery in humans is unknown.

Nursing Mothers: Because of the potential for serious adverse reactions in nursing infants from Wellbutrin, a decision should be made whether to discontinue nursing or to discontinue the drug, taking into account the importance of the drug to the mother.

Pediatric Use: The safety and effectiveness of Wellbutrin in individuals under 18 years old have not been established.

Use in the Elderly: Wellbutrin has not been systematically evaluated in older patients.

ADVERSE REACTIONS: (See also WARNINGS and PRECAUTIONS) Adverse events commonly encountered in patients treated with Wellbutrin are agitation, dry mouth, insomnia, headache/migraine, nausea/vomiting, constipation, and tremor.

Adverse events were sufficiently troublesome to cause discontinuation of Wellbutrin treatment in approximately ten percent of the 2400 patients and volunteers who participated in clinical trials during the product's initial development. The more common events causing discontinuation include neuropsychiatric disturbances (3.0%), primarily agitation and abnormalities in mental status; gastrointestinal disturbances (2.1%), primarily nausea and vomiting; neurological disturbances (1.7%), primarily seizures, headaches, and sleep disturbances; and dermatologic problems (1.4%), primarily rashes. It is important to note, however, that many of these events occurred at doses that exceed the recommended daily dose.

The table below is presented solely to indicate the relative frequency of adverse events reported in representative controlled clinical studies conducted to evaluate the safety and efficacy of Wellbutrin under relatively similar conditions of daily dosage (300-600 mg), setting, and duration (3-4 weeks). The figures cited cannot be used to predict precisely the incidence of untoward events in the course of usual medical practice where patient characteristics and other factors must differ from those which prevailed in the clinical trials. These incidence figures also cannot be compared with those obtained from other clinical studies involving related drug products as each group of drug trials is conducted under a different set of conditions. Finally, it is important to emphasize that the tabulation does not reflect the relative severity and/or clinical importance of the events. A better perspective on the serious adverse events associated with the use of Wellbutrin is provided in the WARNINGS and PRECAUTIONS sections.

TREATMENT EMERGENT ADVERSE EXPERIENCE INCIDENCE IN PLACEBO-CONTROLLED CLINICAL TRIALS* (Percent of Patients Reporting)

Adverse Experience	Wellbutrin Patients (n = 323)	Placebo Patients (n = 185)	Adverse Experience	Wellbutrin Patients (n = 323)	Placebo Patients (n = 185)
CARDIOVASCULAR			Dry Mouth	27.6	18.4
Cardiac Arrhythmias	5.3	4.3	Excessive Sweating	22.3	14.6
Dizziness	22.3	16.2	Headache/Migraine	25.7	22.2
Hypertension	4.3	1.6	Impaired Sleep Quality	4.0	1.6
Hypotension	2.5	2.2	Increased Salivary Flow	3.4	3.8
Palpitations	3.7	2.2	Insomnia	18.6	15.7
Syncope	1.2	0.5	Muscle Spasms	1.9	3.2
Tachycardia	10.8	8.6	Pseudoparkinsonism	1.5	1.6
DERMATOLOGIC			Sedation	19.8	19.5
Pruritus	2.2	0.0	Sensory Disturbance	4.0	3.2
Rash	8.0	6.5	Tremor	21.1	7.6
GASTROINTESTINAL			NEUROPSYCHIATRIC		
Anorexia	18.3	18.4	Agitation	31.9	22.2
Appetite Increase	3.7	2.2	Anxiety	3.1	1.1
Constipation	26.0	17.3	Confusion	8.4	4.9
Diarrhea	6.8	8.6	Decreased Libido	3.1	1.6
Dyspepsia	3.1	2.2	Delusions	1.2	1.1
Nausea/Vomiting	22.9	18.9	Disturbed Concentration	3.1	3.8
Weight Gain	13.6	22.7	Euphoria	1.2	0.5
Weight Loss	23.2	23.2	Hostility	5.5	3.8
GENITOURINARY			NONSPECIFIC		
Impotence	3.4	3.1	Fatigue	5.0	8.6
Menstrual Complaints	4.7	1.1	Fever/Chills	1.2	0.5
Urinary Frequency	2.5	2.2	RESPIRATORY		
Urinary Retention	1.9	2.2	Upper Respiratory Complaints	5.0	11.4
MUSCULOSKELETAL			SPECIAL SENSES		
Arthritis	3.1	2.7	Auditory Disturbance	5.3	3.2
NEUROLOGICAL			Blurred Vision	14.6	10.3
Akathisia	1.5	1.1	Gustatory Disturbance	3.1	1.1
Akinesia/Bradykinesia	8.0	8.6			
Cutaneous Temperature Disturbance	1.9	1.6			

*Events reported by at least 1% of Wellbutrin patients are included.

Other Events Observed During the Development of Wellbutrin: The conditions and duration of exposure to Wellbutrin varied greatly and a substantial proportion of the experience was gained in open and uncontrolled clinical settings. During this experience, numerous adverse events were reported; however, without appropriate controls, it is impossible to determine with certainty which events were or were not caused by Wellbutrin. The following enumeration is organized by organ system and describes events in terms of their relative frequency of reporting in the data base. Events of major clinical importance are also described in the WARNINGS and PRECAUTIONS sections of the labeling.

The following definitions of frequency are used: Frequent adverse events are defined as those occurring in at least 1/100 patients. Infrequent adverse events are those occurring in 1/100 to 1/1000 patients, while rare events are those occurring in less than 1/1000 patients.

Cardiovascular: Frequent was edema; infrequent were chest pain, EKG abnormalities (premature beats and nonspecific ST-T changes), and shortness of breath/dyspnea; rare were flushing, pallor, phlebitis and myocardial infarction.

Dermatologic: Frequent were nonspecific rashes; infrequent were alopecia and dry skin; rare were change in hair color, hirsutism and acne.

Endocrine: Infrequent was gynecostasia; rare were glycosuria and hormone level change. **Gastrointestinal:** Infrequent were dysphagia, thirst disturbance, and liver damage/jaundice; rare were rectal complaints, colitis, G.I. bleeding, intestinal perforation and stomach ulcer.

Genitourinary: Frequent was nocturia; infrequent were vaginal irritation, testicular swelling, urinary tract infection, painful erection, and retarded ejaculation; rare were dysuria, enuresis, urinary incontinence, menopause, ovarian disorder, pelvic infection, cystitis, dyspareunia, and painful ejaculation.

Hematologic/Oncologic: Rare were lymphadenopathy, anemia and pancytopenia.

Musculoskeletal: Rare was musculoskeletal chest pain.

Neurologic: (see WARNINGS) Frequent were ataxia/incoordination, seizure, myoclonus, dyskinesia, and dystonia; infrequent were mydriasis, vertigo, and dysarthria; rare were EEG abnormality, abnormal neurological exam, impaired attention, scintilla and aphasia.

Neuropsychiatric: (see PRECAUTIONS) Frequent were mania/hypomania, increased libido, hallucinations, decrease in sexual function, and depression; infrequent were memory impairment, depersonalization, psychosis, dysphoria, mood instability, paranoia, formal thought disorder, and frigidity; rare was suicidal ideation. **Oral Complaints:** Frequent was stomatitis; infrequent were toothache, bruxism, gum irritation, and oral edema; rare was glossitis.

Respiratory: Infrequent were bronchitis and shortness of breath/dyspnea; rare were epistaxis, rate or rhythm disorder, pneumonia and pulmonary embolism.

Special Senses: Infrequent was visual disturbance; rare was diplopia.

Nonspecific: Frequent were flu-like symptoms; infrequent was nonspecific pain; rare were body odor, surgically related pain, infection, medication reaction and overdose.

Postintroduction Reports: Voluntary reports of adverse events temporally associated with Wellbutrin that have been received since market introduction and which may have no causal relationship with the drug include the following:

Cardiovascular: orthostatic hypotension, third degree heartblock

Gastrointestinal: esophagitis, hepatitis

Hemic and Lymphatic: ecchymosis, leukocytosis, leukopenia

Musculoskeletal: arthralgia, myalgia, muscle rigidity/fever/rhabdomyolysis

Nervous: coma, delirium, dream abnormalities, paresthesia, unmasking of tardive dyskinesia

Skin and Appendages: angioedema, exfoliative dermatitis, urticaria

Special Senses: tinnitus

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MARCH

March 1, symposium, "Treating the Complex Couple in Managed Care Models" (6 hours of CME category I credit available), Harvard Medical School and Harvard Community Health Plan, Boston. Contact: Anne Cronin, Harvard Community Health Plan, Teaching Center, 2 Fenway Plaza, Boston, MA 02215; 617-421-2742.

March 1-3, Sixth Annual Research Conference, "A System of Care for Children's Mental Health: Expanding the Research Base," Research and Training Center for Children's Mental Health, Tampa, Fla. Contact: Dan Casella, 813-974-4433.

March 2–4, annual conference, “The Complex Patient in Time-Effective Treatment” (15 hours of CME category I credit available), Harvard Medical School and Harvard Community Health Plan, Boston. Contact: Anne Cronin, Harvard Community Health Plan, Teaching Center, 2 Fenway Plaza, Boston, MA 02215; 617-421-2742.

March 3–4, symposium, "Pan-American Symposium on AIDS and HIV Disease: A Mental Health Perspective," American Psychiatric Association and the Psychiatric Society of Puerto Rico, San Juan, Puerto Rico. Contact: Christine Dale-Eldridge, AIDS Education Office, APA, 1400 K Street, NW, Washington, DC 20005; 202-682-6147 (tel), 202-682-6114 (fax).

March 5-6, conference, "Addictions," Cambridge Hospital, Harvard Medical School, Boston. Contact: Judy Reiner Platt, Ed.D., Cambridge Hospital, 1493 Cambridge Street, Cambridge, MA 02139; 617-864-6165.

March 8-10, 16th Annual Gold Coast Medicine Conference, "New Frontiers in Primary Care" (10 hours of CME category I credit available), Good Samaritan Medical Center, West Palm Beach, Fla. Contact: Laura J. Keech, Good Samaritan Medical Center, P.O. Box 3166, West Palm Beach, FL 33402-3166; 407-650-6177.

March 10-13, annual meeting, Association for Academic Psychiatry, Charleston, S.C. Contact: Ms. O'Loughlin, Dept. of Psychiatry, Wyman 2, Mount Auburn Hospital, Cambridge, MA 02238; 617-499-5198.

March 18-20, international conference, "1993, European Year of the Elderly: Dysfunction of Mind and Body in the Elderly: Assessment and Intervention." European Office of the World Health Organization, Rotterdam. Contact: S.O.G.G., Mr. R. Kiela or Mr. K. Schilder, P.O. Box 23115, 3001 KC

Rotterdam, The Netherlands;
4367273 (fax).

**March 18-21, 13th National
Anxiety Disorders Association
S.C. Contact: Conference
Rockville, MD 20852; 301-2**

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March 27, conference, "Intensive
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March 29–April 3, course, “Integrative Clinical Neuroscience Update for Neurologists, and Internists,” Massachusetts General Hospital, Harvard Medical School, Boston, Massachusetts (617-726-2000, Neurology, Massachusetts General Hospital, 02114; 617-726-8463).

APRIL

April 1-2, 16th Annual Symposium on Law, Psychiatry and Public Policy
Law, Richmond, Va. Contact:
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INDICATIONS

Treatment of manic episodes of manic-depressive illness. Maintenance therapy prevents or diminishes the intensity of subsequent episodes in those manic-depressive patients with a history of mania.

WARNINGS

Lithium should generally not be given to patients with significant renal or cardiovascular disease, severe debilitation or dehydration, or sodium depletion.

Chronic lithium therapy may be associated with diminution of renal concentrating ability. Such patients should be carefully managed to avoid dehydration with resulting lithium retention and toxicity. Morphologic changes with glomerular and interstitial fibrosis and nephron atrophy have been reported. Morphologic changes have also been seen in manic-depressive patients never exposed to lithium. During lithium therapy, progressive or sudden changes in renal function, even within the normal range, indicate the need for reevaluation of treatment.

An encephalopathic syndrome (characterized by weakness, lethargy, fever, tremulousness and confusion, extrapyramidal symptoms, leukocytosis, elevated serum enzymes, BUN and FBS) has occurred in a few patients treated with lithium plus a neuroleptic. In some instances, the syndrome was followed by irreversible brain damage. Patients receiving such combined therapy should be monitored closely for early evidence of neurologic toxicity and treatment discontinued promptly if such signs appear. Caution patients about activities requiring alertness.

Lithium may prolong the effects of neuromuscular blocking agents. Such agents should be given with caution to patients receiving lithium.

Lithium carbonate may cause fetal harm when administered to a pregnant woman. If a patient becomes pregnant while taking this drug, the patient should be apprised of the potential hazard to the fetus.

Nursing should not be undertaken during lithium therapy except in rare and unusual circumstances.

Not recommended in children under 12.

Elderly patients often require lower lithium dosages to achieve therapeutic serum levels. They may also exhibit adverse reactions at serum levels ordinarily tolerated by younger patients.

PRECAUTIONS

Caution should be used when lithium and diuretics are used concomitantly. Patients receiving such combined therapy should have serum lithium levels monitored closely and the lithium dosage adjusted if necessary.

Sweating, diarrhea and concomitant infection with elevated temperatures may also necessitate a temporary reduction or cessation of medication.

Indomethacin and piroxicam have been reported to increase significantly, steady state plasma lithium levels. There is also some evidence that other nonsteroidal anti-inflammatory agents may have a similar effect. When such combinations are used, increased plasma lithium level monitoring is recommended. Concurrent use of metronidazole with lithium may provoke lithium toxicity due to reduced renal clearance. Monitor patients receiving such combined therapy closely.

When used with angiotensin-converting enzyme inhibitors, such as enalapril and captopril, lithium dosage may need to be decreased; measure plasma lithium levels more often.

Concurrent use of calcium channel blocking agents with lithium may increase the risk of neurotoxicity in the form of ataxia, tremors, nausea, vomiting, diarrhea and/or tinnitus. Caution is recommended.

ADVERSE REACTIONS

Adverse reactions may be encountered at serum lithium levels below 1.5 mEq/L. Mild to moderate adverse reactions may occur at levels from 1.5 to 2.5 mEq/L, and moderate to severe reactions may be seen at levels of 2.0 mEq/L and above. Fine hand tremor, polyuria and mild thirst may occur during initial therapy and may persist throughout treatment. Transient and mild nausea and general discomfort may also appear during initial therapy. These side effects usually subside with continued treatment or a temporary reduction or cessation of dosage. Diarrhea, vomiting, drowsiness, muscular weakness and lack of coordination may be early signs of lithium intoxication, and can occur at lithium levels below 2.0 mEq/L. At higher levels, ataxia, giddiness, tinnitus, blurred vision and a large output of dilute urine may be seen. Serum lithium levels above 3.0 mEq/L may produce a complex clinical picture, involving multiple organs and organ systems. Serum lithium levels should not be permitted to exceed 2.0 mEq/L during the acute treatment phase.

The following reactions appear to be related to serum lithium levels, including levels within the therapeutic range. **Neuromuscular/Central Nervous System**—tremor, muscle hyperirritability (fasciculations, twitching, clonic movements of whole limbs), hyperreflexia, ataxia, choreo-athetoid movements, hyperactive deep tendon reflex, extrapyramidal symptoms including acute dystonia, cogwheel rigidity, blackout spells, epileptiform seizures, slurred speech, dizziness, vertigo, downbeat nystagmus, incontinence of urine or feces, somnolence, psychomotor retardation, restlessness, confusion, stupor, coma, tongue movements, tics, tinnitus, hallucinations, poor memory, slowed intellectual functioning, startled response, worsening of organic brain syndromes; **Cardiovascular**—cardiac arrhythmia, hypotension, peripheral circulatory collapse, bradycardia, sinus node dysfunction with severe bradycardia (which may result in syncope); **Gastrointestinal**—anorexia, nausea, vomiting, diarrhea, gastritis, salivary gland swelling, abdominal pain, excessive salivation, flatulence, indigestion; **Genitourinary**—glycosuria, decreased creatinine clearance, albuminuria, oliguria and symptoms of nephrogenic diabetes insipidus including polyuria, thirst and polydipsia; **Dermatologic**—drying and thinning of hair, alopecia, anesthesia of skin, acne, chronic folliculitis, xerosis cutis, psoriasis or its exacerbation, generalized pruritus with or without rash, cutaneous ulcers, angioedema; **Autonomic**—blurred vision, dry mouth, impotence/sexual dysfunction; **Thyroid Abnormalities**—euthyroid goiter and/or hypothyroidism (including myxedema) accompanied by lower T₃ and T₄ uptake may be elevated. [See PRECAUTIONS.] Paradoxically, rare cases of hyperthyroidism have been reported; **EEG Changes**—diffuse slowing, widening of the frequency spectrum, potentiation and disorganization of background rhythm; **EKG Changes**—reversible flattening, isoelectricity or inversion of T-waves; **Miscellaneous**—fatigue, lethargy, transient scotomata, exophthalmos, dehydration, weight loss, leukocytosis, headache, transient hyperglycemia, hypercalcemia, hyperparathyroidism, excessive weight gain, edematous swelling of ankles or wrists, metallic taste, dysgeusia/taste distortion, salty taste, thirst, swollen lips, tightness in chest, swollen and/or painful joints, fever, polyarthralgia, dental caries. Some reports of nephrogenic diabetes insipidus, hyperparathyroidism and hypothyroidism which persist after lithium discontinuation have been received.

A few reports have been received of the development of painful discoloration of fingers and toes and coldness of the extremities within one day of the starting of treatment with lithium. The mechanism through which these symptoms (resembling Raynaud's syndrome) developed is not known. Recovery followed discontinuance.

Cases of pseudotumor cerebri (increased intracranial pressure and papilledema) have been reported with lithium use. Lithium should be discontinued, if clinically possible, if this syndrome occurs.

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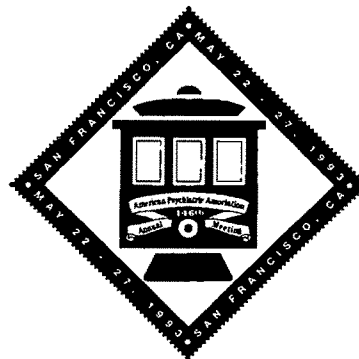
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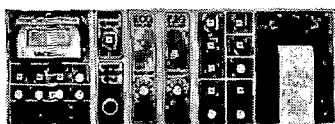
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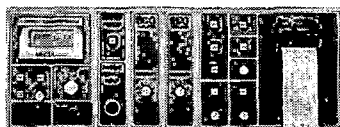
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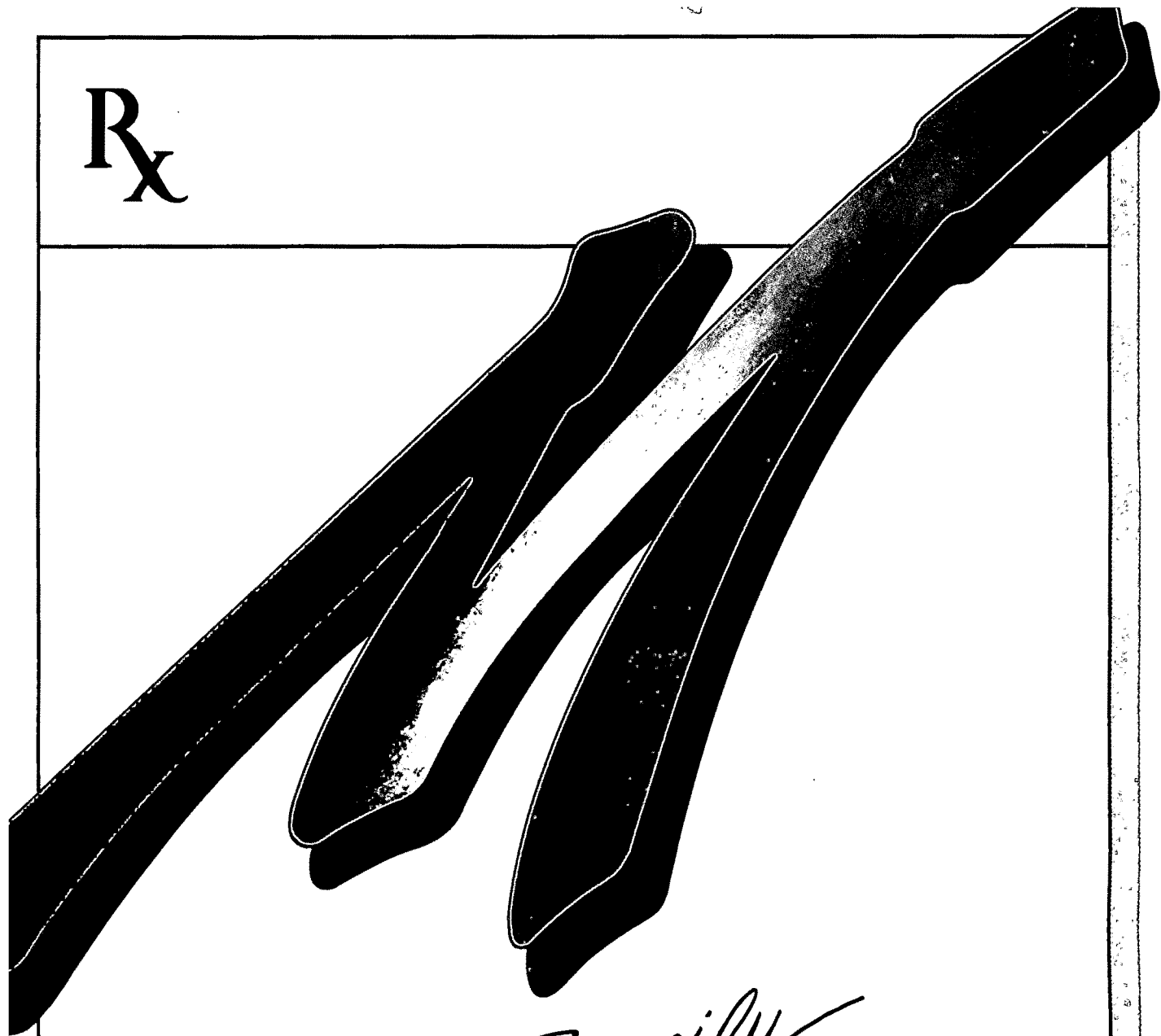
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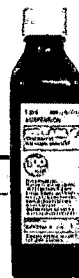
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New Year's Greetings

"A man had neede of long-tough eares," commented Montaigne in his essay "Of Experience," "to heare himself freely judged. And because there can be few that can endure to heare it without tingling: those which adventure to undertake it with us, shew us a singular effect of true friendship. For, that is a truly perfect love, which to profit and doe good, feareth not to hurt or offend."

Montaigne's concern, of course, was with the more general question of the profit and peril inherent in offering constructive criticism, but his words speak pointedly across the centuries to a modern editor, his authors, and his reviewers, whose critical comments about manuscripts submitted to the *Journal* are so central to maintaining its scientific and literary standards. In our nearly fifteen years as the *Journal's* Editor, we have never ceased to be amazed how our authors' "long-tough eares" have enabled them, however much they may have "tingled," graciously to accept and incorporate into their writings the criticisms and suggested revisions deriving from peer review. And even more, we shall remain ever indebted to our myriad reviewers, who in "true friendship" have almost invariably been willing "to profit and doe good" to their scientific colleagues and to our profession as a whole. It gives us great pleasure, therefore, to acknowledge our gratitude to the following men and women who have given us their editorial advice and counsel during the period from November 1, 1991 to October 31, 1992.

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As he nears the end of his term of office, the Editor must express his deep satisfaction with the warm and lasting relationships he has enjoyed with his editorial companions. The skill and friendship of his Deputy Editor, Dr. Nancy C. Andreasen, has immeasurably lightened the burden of his task, and his Associate Editors have invariably responded with the help and advice he has needed. Although he came into office a statistical ignoramus who thought Bonferroni an Italian sports car, his Statistical Editors, Drs. John J. Bartko and Lee Gurel, have patiently taught him the basic elements of statistical science while ensuring the proper use of statistical methods in the *Journal's* pages. And without the congenial and devoted collaboration of Sandra Patterson, his Managing Editor, and her entire editorial staff, there would be no *Journal* at all.

Throughout the Editor's tenure, in good times and bad, he has enjoyed a long and rewarding friendship with the Medical Director, Melvin Sabshin, whose support and encouragement have always been matched by the Officers and Board of Trustees of the Association. And for their help in innumerable ways, he must also extend his sincere appreciation to Drs. Robert J. Campbell III, William A. More, Harold A. Pincus, Carolyn B. Robinowitz, John A. Talbott, and Jack W. White; to Mses. Laura Abedi, Mary Ellen Celik, Teddye Clayton, Carol Davis, Elizabeth Flynn, Nancy Frey, Debbie Goldberg, Alison Jones, JoAnn Macbeth, Beth Prester, and Jacqueline Young; and to Messrs. William E. Baxter, John Blamphin, George Campbell, Herbert M. Gant, Ronald McMillen, Raymond J. Purkis, Jr., and Michael Roy.

As the new year dawns and we sail the last leg of our editorial journey toward home port, we wish our many colleagues and readers a happy, prosperous, and literate 1993. Hail and farewell!

J.C.N.

Psychiatric Care and Health Insurance Reform

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Concerns about cost, access, and quality of health care in the United States have led to a variety of legislative proposals that would reform our health care system and its financing. Health insurance benefits for mental illness, including substance abuse, are treated differently from medical/surgical benefits, with stricter limits on outpatient visits and hospital days. Medicare, Medicaid, and most private health insurance plans contain this historic disparity of coverage for mental illness compared to general medical illness. Psychiatric services are also distinguishable because of the large public sector reimbursement for mental illness treatment and support. Principles for a more equitable design of mental health benefits include a non-discriminatory approach; payment on the basis of service rather than diagnosis; application of cost containment for care of mental illness on the same basis as care of general medical illness; retention of the public sector as a backup system for high-cost, long-term care; encouragement of lower-cost alternatives to the hospital through the development of a continuum of care; and a recognition of the distinction between psychotherapy and medical management. All current approaches to universal health care fall short of these principles. A research agenda is needed now more than ever in order to articulate the case for complete coverage of mental illness and substance abuse.

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National health insurance has had a turbulent history since the issue was first proposed in the United States in the late 1930s. Legislation in numerous forms has been proposed regularly from 1939 (1) to the present time, but a comprehensive national program has never achieved passage. The 1965 Medicare and Medicaid programs were partial solutions created out of political compromise.

In the mid-1970s, proposals for national health insurance were supported by Presidents Nixon, Ford, and Carter, as well as by leading congressional Democrats, notably Senator Kennedy of Massachusetts. However, none of these proposals achieved passage and it seemed

once again that universal care through a national health insurance system was an idea that had come and gone.

Today, however, growing concerns about cost, access, and quality within our health care system compel a reanalysis of the fragmented legislative approaches that have historically been taken toward these problems. A nationwide debate is again underway about the need for fundamental reform of the system in order to provide universal access to medical care through the creation of a national health insurance program. The question has come up whether this should take the form of a single *national* health insurance program that covers all Americans, or a *universal* health insurance program that provides coverage to those currently uninsured but leaves intact the current multiplicity of public and private plans. It is generally agreed that any new system must concurrently deal with access and cost issues.

This paper will review some of the background that generates the need for such a reanalysis of U.S. health care financing and examine some of the issues arising

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from health insurance reform. Some universal health insurance policy issues would affect all physicians, while others confront psychiatrists uniquely. We then focus on the specific problem of benefits for mental illness, including substance abuse, under universal health insurance, the implementation of which creates critical policy issues. These issues include the historic disparity in coverage between psychiatric and other conditions, the distinction between medical management and psychotherapy, and the incentives for a continuum of care. Approaches to coverage for mental disorders in current universal health insurance proposals are very narrow, representing yet more patchwork solutions to major gaps in coverage.

We do not attempt to determine whether or not it is time to implement universal coverage. We do not evaluate every aspect of the various proposals, such as their system-wide financing strategies. Nor will we examine every issue relevant to mental illness benefits under universal insurance.

We do explore how these approaches to universal health insurance would affect care for mental disorders. We compare and contrast these proposals to psychiatric benefits in the major current insurance programs and to a new model mental health benefit. We then examine policy issues that face our profession and legislators, delineating strategies for mental health coverage under universal health care. Finally, we provide a commentary on gaps in our knowledge base regarding the effectiveness and costs of treatment for mental illness. We believe this research agenda should be pursued now in anticipation of major reform in the U.S. health care system.

COVERAGE OF PSYCHIATRIC CARE IN TODAY'S SYSTEM

Private Sector

The purpose of all casualty insurance is to provide protection against catastrophic financial loss. The principle of health insurance is to provide financial protection against medical expenses to consumers in return for a fixed, predetermined premium (2). Insurance companies do not intend to provide coverage for all medical care, as it is an expected expense. Their goal is (or should be) to insure against catastrophic economic loss (which could be arbitrarily defined, as in the tax code, as 7.5% of annual adjusted gross income) due to medical expenses. Today's insurance consumers desire expanded coverage, elimination of exclusions for preexisting conditions, and implementation of community-wide actuarial risk for determining premium prices. Each of these modifications would increase premium prices and therefore conflict with the cost-containment agenda of employers, public sector insurers, and the insurance industry (3). Improved access to medical care for the covered population is an added, societal benefit of health insurance. However, budget

constraints create tension between these various goods: given dollar limits, trade-offs are inevitable.

Historically, care for mental disorders has received insurance benefit treatment separate from the rest of medical care. This is due to several factors, including ambiguous descriptions and classifications of mental disorders, misunderstanding of the types of care provided for mental disorders, perception that liberalization of benefits would unduly increase demand for services, and the stigma attached to mental illness, substance abuse, and their treatment. In addition, it has historically been the *states'* responsibility to provide psychiatric services directly to the indigent without insurance and to the insured whose benefits have run out.

Benefits for mental disorders and substance abuse in the private sector vary widely. Although most health insurance policies have some coverage for treatment of mental illness, the vast majority have *inside limits* in which psychiatric care is treated differently from medical-surgical care (4-6). Inside limits include higher cost sharing and limits on outpatient visits or hospital days. These limits generally result in noncoverage of catastrophic expenses while providing a partial subsidy for expenditures up to an arbitrary limit. The patient then pays out of pocket, forgoes care, or, in the case of severe impairment, may seek care in the public sector. Insurance therefore has failed the mentally ill as a protection against catastrophic economic loss.

The costs of medical care are a great impetus to reform and underlie many of the proposals for universal health care. Reports of disproportionate increases in costs of mental health and substance abuse care under private insurance have appeared in the media in recent years. The benefit consulting firm A. Foster Higgins has cited increases far in excess of general medical increases during the years 1987 to 1989. Recent studies by Frank and associates (7, 8) examined the claims experience of employees of mid-size to large U.S. manufacturing firms between the years 1986 and 1989. They found that the growth of charges for inpatient and outpatient substance abuse treatment, and, although supportive data are less conclusive, an increase in adolescent inpatient treatment from the years 1986 to 1988 contributed most strongly to the disproportionate growth of mental health and substance abuse expenditures. In fact, costs of adult inpatient psychiatric treatment grew less rapidly than costs for all inpatient care. These data also indicated that increases in mental health charges contributed only 2.5% of the 13% increase in global medical charges between 1986 and 1989. After substantial cutbacks in coverage for mental health and substance abuse in 1988 and 1989, the growth in *overall* charges was not substantially affected: they continued to rise at a rate of close to 12%. These studies support the idea that differences exist within the broad category of treatment of mental disorders and substance abuse, and consideration should be given to design of insurance benefits that recognize those differences.

In the cost-conscious 1980s, many companies have further reduced the inside limits for the treatment of

mental disorders, eroding benefits in the private sector. In 1986, for all participants receiving psychiatric benefits under private sector coverage, 99% had inpatient coverage and 97% had outpatient coverage, but only 37% of inpatient and 6% of outpatient coverage were at parity with coverage for other illnesses (9). Inside limits for the care of mental disorders heighten the underinsurance of these conditions for the population.

Employers have implemented numerous additional strategies designed to reduce costs. "Carve-outs," in which mental illness and substance abuse benefits are treated differently from the rest of the policy, are common. Carve-outs may include contracting benefits to an independent company or a mental health subsidiary of an insurance company. They may include internal management of mental illness through an employee assistance program, case managers, or other gatekeepers to the mental health system. Carve-outs may affect choice of provider, whereby companies use preferred provider networks or contract directly with local providers (10).

Managed care provides a major regulatory review of the appropriateness of services provided to beneficiaries. The goal of managed care is to reduce costs and regulate access to care. Not surprisingly, psychiatric care is often managed differently from other medical-surgical care. A survey of 145 organizations in the late 1980s found that over 70% already had managed care for psychiatric treatment or were considering implementing it: this was a 10% increase from results of similar surveys conducted 3–4 years previously (11).

Mandated minimum benefits, legislated in 28 states between 1971 and 1988, are aimed at providing a basic minimum of care for mental disorders and/or substance abuse. Mandates spread the risk of catastrophic losses and therefore expand the potential for access to coverage even as benefit limits and managed care constrain this access. Mandates are an effort to overcome "adverse selection" (12) (i.e., those most likely to use a specific benefit attempt to purchase a policy providing coverage of it, driving up premium prices) and failure of the market to provide health insurance coverage for mental disorders and substance abuse. There have been problems, however, with this approach for small employers who cannot afford the premiums and a further segmenting of the market as more companies self-insure or turn to health maintenance organizations (HMOs), which in many states are exempt from the mandates.

Mandated minimum benefit requirements vary widely among states, ranging from parity with medical-surgical illnesses to very limited coverage (9). Sixteen states mandate minimum benefit packages, and another 12 mandate availability of certain types of coverage for mental illness. Drug abuse treatment is covered by nearly half, and alcoholism treatment is covered by all but one of the mandates (13).

Many employees receive health insurance as a fringe benefit of employment. However, small businesses have great difficulty affording insurance premiums for their employees. Those who work do not qualify for public assistance programs. Further exacerbating the prob-

lem, individual insurance policies are unaffordable for most Americans because they are unable to negotiate favorable rates like large employers. Thus low-wage workers and small business employees are particularly likely to be uninsured. The Robert Wood Johnson Foundation estimates that more than three-fourths of uninsured Americans are either workers or dependents of workers (14).

Because they are perceived as expensive components of health insurance policies, the mandates have been blamed by some for the underinsurance of employed Americans. Recent legislative trends are to reduce or eliminate them. Bare-bones legislation, permitting insurance companies to market minimum benefit policies that exclude coverage of mental disorders and substance abuse, has passed in 12 states (3, 15). These laws facilitate the purchase of insurance policies by individuals and small businesses by allowing minimum-coverage policies that are theoretically less expensive, thereby expanding access. In fact, however, most cost savings (40%–50% reduction in average premium costs) are not attributable to savings from elimination of mandates but from increased cost-sharing provisions (3).

Although HMOs are also part of the private sector, different regulations apply to them (8, 16–36) (table 1). HMOs are often exempt from state mandates. Although on average they are more restrictive of inpatient psychiatric coverage, HMOs have inpatient substance abuse coverage more often than do other insured or self-insured plans (9).

Further exacerbating the underinsurance of mental disorders and substance abuse, companies that self-insure are also exempt from the mandates. In 1988, nearly 60% of Americans covered by conventional group insurance were enrolled in a plan that was at least partially self-insured (13). As more Americans come under self-insurance or become uninsured, and as bare-bones legislation is implemented in more states, the mandates are having less of an impact on access than was hoped.

Public Sector

The public sector reimburses a greater percentage of psychiatric care in organized settings than does the private sector. In 1986, 70% of revenue from mental health organizations derived from public sources, with 30% from patient fees and other sources (13). The public sector encompasses Medicare, Medicaid, the Indian Health Service, and the Veterans Administration system at the federal level; Medicaid and public hospitals at the state level; and local systems.

The Medicare program will be described in more detail because it is the foundation for several of the universal health proposals described later in this article and could be the framework for implementation of benefits under a universal health insurance plan. Medicare is a form of universal health coverage for all people age 65 and over who are eligible for Social Security and for

TABLE 1. Proposals for Health Insurance Reform^a

Sponsor	Proposal	Mental Health Benefit
S. 1177: Pepper Commission on Comprehensive Health Care (Rockefeller Bill) and H.R. 2535 (Waxman Bill)	Mandates employer-provided health insurance, eliminates Medicaid, implements public health care plan for the poor and unemployed	45 inpatient days per year with 20% copayment, 25 outpatient visits per year with 50% copayment
H.R. 650: MediPlan Act (Stark Bill)	Covers all Americans with expanded Medicare benefits. Elderly would continue to be covered by Medicare	Same as Medicare benefits, with 50% copayment for outpatient visits
H.R. 1300: Universal Health Care Act (Russo et al. Bill)	Single-payer system with national fee schedules	45 inpatient days per year, 20 outpatient visits per year, no deductibles or copayments
H.R. 1565 (Chandler Bill)	Requires all insurance companies to offer basic benefit plan to small businesses. Plans exempt from both state mandates and state limitations on utilization review and managed care	No mental illness benefits
S. 700 (Durenberger Bill)	Requires insurers to offer employers a basic benefit plan. State mandates exempted	No mental illness or substance abuse coverage in core benefit. Upgraded standard plan covers 30 days of inpatient care per year and 25 outpatient visits per year, both with 50% copayment
H.R. 1255 (Pease Bill)	National health insurance program for the uninsured. Financed by increased cigarette tax	Inpatient and outpatient services by physicians, <i>presumably</i> including psychiatrists
H.R. 2114 (Sabo Bill)	Requires states to develop health insurance plans to be made available to all state residents	Inpatient services and prescription drugs covered; no outpatient benefits
S. 1227: "Health America": Affordable Health Care for All Americans (Kennedy-Mitchell Bill)	Universal coverage and cost-control program. Requires all employers to provide basic health insurance. Replaces Medicaid with new program, "American care" (state-administered, financed by payroll tax)	45 inpatient days per year; 20 outpatient visits per year with 50% copayment; includes reimbursement for care by nonphysicians; physician visits for medical management with 20% copayment; covers partial hospitalization and residential care
H.R. 3205: Health Insurance Coverage and Cost Containment Act of 1991 (Rostenkowski Bill)	Mandates employer-provided health insurance or contribution to public health insurance plan. Public plan modeled on Medicare with pediatric, preventive, and obstetric services. Includes expenditure targets and rate-setting of reimbursement levels	Public plan benefits same as Medicare. Mental health benefits in employer plans not specified. \$250 individual deductible, \$500 family deductible with catastrophic limit on out-of-pocket expenses
H.R. 2530 (Sanders Bill)	State-administered program. Comprehensive benefits based on general federal guidelines	Not specified. Intended as medically necessary with full coverage, as for other medical care
"U.S. Health Act" (Roybal Bill)	Universal enrollment for catastrophic, long-term, and basic benefits. Consolidates Medicare, Medicaid, and private insurance into single system. Funded by taxes and beneficiary cost sharing. Includes cap on national expenditures	45 inpatient days per year with 20% copayment; 25 outpatient visits per year with 50% copayment; modifications in progress
"Health Security Partnership" (Committee for National Health Insurance)	Federally determined core benefit plan, administered by states. Funded by premiums and state and federal taxes. Includes deductibles and coinsurance	45 inpatient days per year without copayment, 20 outpatient visits per year without copayment, 15 additional outpatient visits per year with copayment
Davis Proposal	Requires employer-provided coverage. States have buy-in option to Medicare for Medicaid enrollees. Remaining uninsured enrolled in Medicare with income-related premiums. Has deductibles and coinsurance with out-of-pocket annual limits	Same as Medicare benefits
Health Access America (American Medical Association Proposal)	16-point plan, including Medicare and Medicaid reform, required employer provision of health insurance, change in tax treatment, creation of state risk pool, liability reform, and practice parameters	Detoxification not covered; 20 outpatient visits (all types) per year; 45 inpatient days (all types) per year; \$350 deductible (individual) or \$750 deductible (family) plus copayments
Consumer Choice Health Plan	Managed competition strategy with public sponsors of private-sector health care financing and delivery, mandated employer-sponsored insurance. No initial change in Medicare or Medicaid	Plans must include basic benefits in HMO Act ^b , possibly with tighter restrictions to reduce costs
Heritage Foundation Proposal	Restructuring tax incentives with refundable tax credits for purchase of insurance. Medicare and Medicaid beneficiaries receive vouchers to purchase insurance	Varies depending on plan; supports repeal of state-mandated benefits
Physicians for a National Health Program	Universal health insurance with single government payer	In progress
Physicians Who Care Plan	Universal minimum-benefit plan provided by employers. Pre-tax employee accounts as a form of self-insurance. Expanded Medicaid eligibility. Has high deductible (\$1,000). Incorporates scientific medical care guidelines	Mental health benefits not anticipated at this time; detoxification not covered; would eliminate state-mandated benefits

TAB_E 1 (continued)

Sponsor	Proposal	Mental Health Benefit
President Bush's Health Care Reform Proposal	Requires insurers to cover all groups seeking insurance. Eliminates preexisting exclusions. Facilitates small business risk pooling. Incorporates standardized claim forms and electronic billing. Tax credits or deductions to purchase insurance, with vouchers for low-income persons	Not specified. Would eliminate state-mandated benefits
Model Mental Health Benefit by Frank et al. (8)	Model plan for private health insurance benefit design	Unlimited inpatient, day hospital, and other residential care after deductible met, equal to 1 day's cost at facility per episode. Facility-based professional services reimbursed on same basis as other medical benefits. Outpatient psychotherapy—unlimited number of visits with 50% copayment. Outpatient medication management—unlimited number of visits with 20% copayment

^aData are from references 8 and 16–36.

^bThe HMO Act requires short-term (up to 20 visits) outpatient evaluation and crisis intervention mental health services, medical treatment, and referral services for alcohol and drug abuse or addiction. A 1976 amendment stipulated that HMOs may offer supplemental mental health services not included in the basic benefits (37).

those under age 65 who have been receiving Social Security disability payments for at least 2 years. All eligible people are enrolled in Part A (hospital insurance) and may voluntarily enroll in Part B (supplemental medical insurance) by paying a premium deducted from their Social Security payment. For a number of reasons, including skyrocketing health care costs, Medicare now pays less than one-half the costs for people over age 65 (16).

Part A, which imposes a lifetime limit of 190 days in free-standing psychiatric hospitals, was legislated to ensure that Medicare would not pay for long-term custodial support of the mentally ill in public hospitals. There is no limit on the number of days covered for the treatment of mental disorders in general hospitals, although hospital coverage for all conditions is limited to 90 days in a benefit period, with the first day's payment as a deductible. (The benefit period begins with the beneficiary's first day of hospitalization and ends when the beneficiary has not been in a hospital or a skilled nursing facility for at least 60 consecutive days.) For Medicare Part B, 80% of approved charges for general medical conditions are paid after a deductible is met. Coverage for physician services provided in inpatient settings is the same for psychiatric and other conditions (although the recently introduced Medicare Fee Schedule severely limits psychiatric fees). In outpatient settings, however, mental illness treatment benefits are more complex.

Medicare always paid for the *evaluation* of mental disorders on the same basis as the evaluation of all general medical conditions. Treatment, however, was subject to inside limits. Since its passage in 1965, Medicare had paid a maximum of \$250 (50% of approved charges up to \$500) for the treatment of mental disorders. In 1987, the program's mental health coverage was expanded from \$250 to \$1,100, or from 50% of \$500 to 50% of \$2,200. The 1987 modifications also exempted payment for the *medical management* of mental disorders from these restrictions and covered it on the same basis as all other conditions, that is, 80%

of approved charges. Medical management of mental illness includes prescribing, monitoring, and changing prescription drugs used in the treatment of mental disorders. A partial hospitalization benefit was also added in 1987 to expand coverage in outpatient settings. In 1989 the special dollar limit (\$1,100) on payments for psychotherapy was removed, although 50% cost sharing was retained. Also in 1989, independent provider status was extended to psychologists and social workers (38).

Medicaid is the federal-state program legislated in 1965 to provide medical care for means-tested indigent persons in the United States. States are permitted to design their own programs under broad federal guidelines. Mandatory benefits under Medicaid for the treatment of mental disorders include inpatient care in general hospital psychiatric units, outpatient care in general hospitals or qualifying psychiatric hospitals, day care, night care and partial hospitalization when associated with outpatient hospital service, and physician services, although visit limits may be set. Care in institutions for mental disease (i.e., facilities that care for a population in which more than 50% of the patients have a primary psychiatric diagnosis) is specifically excluded for beneficiaries from ages 22 to 64. Care in skilled nursing facilities for individuals over age 21 is provided for those with psychiatric illness with an accompanying physical illness and for those with Alzheimer's disease. Optional coverage includes inpatient psychiatric care in institutions for mental disease for beneficiaries over age 65 and under age 22, free-standing clinics (including community mental health centers), day care, night care and partial hospitalization associated with free-standing clinics, and independent reimbursement of clinical psychologists and social workers (9). Facilities including halfway houses, adult residential foster homes, and crisis centers do not qualify for Medicaid reimbursement (39).

Trends in the Medicaid program have been toward reduction of eligibility levels and mandatory coverage

over time (40). In 1986, the program covered only 41% of Americans below the poverty level (41).

As a final major component of the public sector, state mental health programs have a 100-plus-year tradition of providing care, primarily for those with severe and persistent mental illness. The state system is also intended as a safety net for those without insurance coverage for private sector care, protecting against what are often catastrophic costs for inpatient psychiatric hospitalization.

However, the public and private sectors do not provide adequate coverage for the U.S. population. It is now estimated that 37 million Americans have no health care insurance. In addition, 15 to 30 million more Americans have inadequate insurance, particularly with respect to benefits for mental disorders (42, 43). Thus, despite massive cost increases, access to care and risk of catastrophic medical expenses are major problems in the U.S. mental health care system. Private sector insurance trends toward managed care, bare-bones policies, and other cost-containment strategies serve to further reduce access to care for mental disorders. Public sector budget constraints exacerbate the problem.

In the treatment of mental illness and substance abuse we have the most dramatic example of our two-class system of medical care—one for the well-insured and employed population, another for the underinsured and uninsured. Universal health insurance could reduce the disparity between these groups, as well as provide incentives for expanding access, containing costs, and improving quality.

THE ROLE OF MENTAL HEALTH COVERAGE

In 1976, Astrachan et al. identified four major tasks of psychiatric care: medical, reparative, social control, and humanistic (44). We present these tasks as a basis for a discussion of the likely effect of national health insurance on each of them. The psychiatrist's medical tasks include differential diagnosis and treatment of illness (both curative and palliative). Reparative tasks attempt to assist patients in compensating for their particular difficulties. Chronically psychotic patients, persons with mental retardation, and patients with severe symptoms of a number of other psychiatric conditions commonly require this mode of care. The third psychiatric task involves social control: certain behaviors are identified as deviant or unacceptable, and providers are expected to attempt to modify patient behaviors. The fourth area of psychiatric tasks encompasses the humanistic realm. In this role, providers assist patients in their desire for personal growth, improved problem-solving ability, and increased self-understanding (44). Under the present U.S. health care system, medical tasks alone are significantly covered. An exception is Medicaid, which covers some nonacute services, including nursing home care, physical rehabilitation, and preventive services.

Insurance coverage of psychiatry's medical tasks usu-

ally includes coverage of acute treatment of mental illnesses through short-term hospitalization and a limited number of annual outpatient visits (the latter usually require a significant copayment). Current policies are often inadequate even for "acute" treatment in the sense that many conditions require treatment beyond the 30 days typically covered. These conditions also frequently require regular office visits for medication management, monitoring mental status, and optimizing treatment compliance. Patients and their families are often subject to high medical bills after they reach insurance coverage limits for both inpatient and outpatient treatment.

BASIC BENEFITS VERSUS CATASTROPHIC COVERAGE

In general insurance parlance, "access" means reducing the financial barriers to use of care, especially initially (i.e., increasing the probability of use). A policy that increases access may also ultimately restrict overall use (i.e., quantity of use), and fail to protect beneficiaries, if limits are imposed on "high use."

There are two basic approaches to covering the costs of the medical tasks of psychiatry, a polarity reflected in the options for benefit design under national health insurance. These approaches have different effects on access to services. The first comprises access to comprehensive coverage with protection against catastrophic financial loss (including those due to long-term care needs). This approach theoretically makes the "public sector," charity care, hospital bad debt, and indigent risk pools things of the past. Financial barriers to access to care are minimized under such arrangements.

The second approach is a basic benefits policy designed to provide access to limited care for everyone but not to offer protection against catastrophic losses. This approach follows the same tactic as mandated benefits in that it defines universal services and reduces the effects of adverse selection, but limits benefits. Basic benefits are also less costly than comprehensive coverage. The basic benefit plan gives everyone access but does not give everyone coverage for sufficient services to meet the full range of needs for care and treatment. In order to deliver appropriate services to everyone, the basic benefit requires any or all of the catastrophic protection mechanisms for beneficiaries or providers: public or charity hospitals, bad debt, or reinsurance risk pools.

Frank and associates (8) have developed a model mental health benefit plan for the privately insured, employed population based on insurance principles and the accumulated body of mental health services research. Its approach is intermediate between comprehensive national health insurance and basic benefits. It was designed to offer catastrophic acute coverage at a reasonable cost, limiting care by judiciously applying the most refined and appropriate cost-containment techniques available. As a compromise between the two approaches, it recognizes the existence of the public sec-

tor for the uninsured but attempts to forestall its use by insured individuals (who may be underinsured for mental health care) through a variety of mechanisms to reduce the provider's incentive to "dump" the patient and to protect beneficiaries from financial risk.

The model mental health benefit is designed for use by employers but is applicable to a universal coverage system. In this plan, hospital, residential, and partial hospital facility care would be fully covered after a deductible (equal to 1 day's cost at the facility) was met; facility-based professional services would be covered at parity with other medical services; outpatient psychotherapy would be covered with 50% copayment with no limit; and outpatient medication management would be covered at parity with other medical visits with 20% copayment.

PRINCIPLES FOR DESIGN OF MENTAL HEALTH BENEFITS

Passage of universal health insurance legislation may not be imminent. However, the large number of proposals and the extent of debate make it worthwhile for psychiatrists to face now the potential clinical and policy consequences of such legislation. Recommendations can be developed now for an approach to psychiatric care under a modified system, anticipating what may be inevitable reform. In this paper, we hope to derive principles for an approach to optimal benefit design for the treatment of mental disorders and substance abuse.

Nondiscriminatory Coverage

Fayers and policy makers seem to vacillate between extreme views of those who seek treatment in the mental health sector. On one hand are those who are hopelessly mentally ill, for whom care is perceived to be futile. On the other hand are the "worried well," a term sometimes applied pejoratively to those whose care is perceived to be discretionary. Psychiatric care itself is misunderstood, seems ill-defined, and remains stigmatized. Furthermore, mental health care has traditionally been a public responsibility. These reasons have historically bolstered its separate insurance treatment (6).

However, there are powerful reasons to conquer this discrimination, including the remedicalization of psychiatry, the development of effective treatments, and the similarities between psychiatric and general medical care. In addition are the societal benefits accruing from psychiatric care, concurrent with significant unmet demand for it. Now is the time to make a case for the elimination of discriminatory policies. Any universal health insurance proposal should be nondiscriminatory with regard to psychiatric benefits.

Pay on the Basis of Services—Not Diagnosis

Psychiatric care is analogous in many ways to general medical care: it encompasses inpatient treatment (of

acute illness and exacerbation of persistent illness) and outpatient visits (for medical management, support and encouragement, and monitoring compliance). Psychiatry has few procedures—namely ECT, psychoanalysis, and psychotherapy—but their use is based on clinical indications and diagnosis, analogous to medical and surgical procedures (45).

For all other conditions, limits are based on the type of treatment and services provided, not on diagnosis. Few, if any, other insurance benefits are based on diagnostic category alone. Furthermore, data do not support the use of such insurance approaches: studies have shown that hospital resource use is not predicted by patients' psychiatric diagnosis (46). For example, knowing that a patient has a diagnosis of schizophrenia does *not* indicate how long hospitalization is likely to be—there is no typical length of stay.

Working for the most part without benefit of data, insurers have assumed that all psychiatric benefits were comparable. For example, because of the greater price sensitivity for *some* types of ambulatory care (e.g., psychotherapy), *all* outpatient treatments have been covered in the same fashion, with greater cost-sharing (47). However, study of ambulatory psychiatric services indicates that not all services are the same (48).

Benefits approaching parity for psychiatric care have been implemented in legislative modifications of the Medicare program. Medicare benefits over time for outpatient treatment have been continually increased, tending toward nondiscriminatory coverage. Psychiatric care could be covered in a fashion similar to other conditions in the design of a future universal insurance system.

Apply Cost-Containment Principles Identically

In most private policies, annual or lifetime limits are placed on the number of inpatient days for beneficiaries hospitalized for psychiatric disorders. Limits are also placed on the annual number of outpatient visits for the treatment of mental disorders and substance abuse. Should psychiatric patients be treated differently from those hospitalized with all other disorders? In private insurance, no other condition evokes day and visit limits to the same extent as mental disorders and substance abuse.

The different treatment of mental disorders and substance abuse has been justified by insurance companies by the increased elasticity of demand for psychiatric services. Elasticity of demand represents the graphic relationship between cost of a service and consumers' demand for the service. Elasticity indicates that demand is sensitive to product price, that is, as the prices of services rise or fall, demand changes concomitantly. Insurance lowers the price of care to the patient. We say demand is inelastic when service utilization is not affected very much by changes in price. Mental health services are more elastic in the aggregate than general health services (49). The use of aggregate data, however, masks variation between different types of psychiatric serv-

ices, some of which may be very sensitive to price (e.g., psychotherapy), while others may be comparatively inelastic (e.g., medical management).

At times cost-containment techniques produce short-term savings but shift costs to other payers or to a later date. When length of stay is reduced by coverage limits, readmission rates increase, as do state mental hospital transfers. Both of these results erode cost savings and concomitantly could lead to poorer patient outcome and quality of care (50). These results also suggest that necessary care is being given, in that private sector insurance limits simply shift the funding source to patients or the public sector.

An equitable system will encourage access as much as possible without sacrificing catastrophic coverage. Any universal health insurance system should apply cost-containment principles identically in a nondiscriminative fashion to psychiatric care and other medical care. Psychiatric care is responsive to supply- and demand-side economic forces, and the system would be improved by ending discrimination by diagnosis in the application of cost-containment measures. For example, we recommend that insurers or managed care organizations review *all* outpatient claims regardless of diagnosis after 20 visits or subject all inpatient stays to length-of-stay limits.

Retain the Public Sector as a Backup System for High-Cost, Long-Term Cases

For a number of reasons, the public sector has traditionally provided a greater proportion of the care for mental disorders and substance abuse than for other conditions. It is the catastrophic care system for underinsured and uninsured Americans. A number of factors in the present system, many of which would likely continue even under future reform, generate the need for a public system of care. Some patients never get well; many suffer from progressive deterioration leading to extreme impairment. Often such patients never become independent of psychiatric support (6). Sharfstein (51) quoted H.H. Goldman and R.G. Frank's statement at a 1985 meeting that

Treatment for this group of patients is very expensive. The direct costs of care for persons suffering chronic mental disability was estimated at \$7.4 billion in 1980, of which more than half was spent in state mental hospitals (\$2.3 billion) and nursing homes (\$1.7 billion). The sum represented 43% of direct costs for all mental health care that year, yet it was generated in caring for only 7% of the country's mentally ill population.

In 1980, less than 10% of the mentally ill used more than one-third of treatment costs (39), a trend that likely continued in the 1980s.

Treatment of severe and persistent conditions and long-term care are a major public health crisis in the United States today. As noted earlier, the public systems are in the process of progressive failure and need basic reform and additional financing (52).

Encourage Lower-Cost Alternatives to the Hospital and a Continuum of Care

Not every patient symptomatic with a mental disorder or substance abuse needs the protection or supervision available in 24-hour inpatient care. Just as an exacerbation of diabetes mellitus or chronic obstructive pulmonary disease can often be managed with frequent outpatient visits and intensified medication management, many psychiatric patients with acute exacerbations could be treated with day hospitalization to adjust medication, more frequent office visits, domiciliary care, or in other outpatient treatment settings. However, insurers have not traditionally recognized the differences among various outpatient settings, nor have they structured benefits to encourage use of such alternatives. As a result, in-hospital stays are longer than necessary. Because this is the most expensive treatment setting, opportunities for cost savings are lost. Alternative economic incentives can promote care in other settings by limiting hospital care, providing continuity of care, providing easy access to care without initial financial barriers to diagnosis and treatment, and introducing consumer and provider options for flexibility in treatment decisions (6). General medicine has had a strong move to alternative outpatient settings, with surgi-centers, ambulatory care centers, convalescent care units, and so forth.

Numerous studies have shown the cost effectiveness of alternatives to hospitalization. For example, a study published in 1990 compared the effects within a group of mentally ill individuals of providing 200 days of halfway house care as part of insurance coverage versus no benefit beyond hospitalization. "Yearly recidivism rates fell from 79% to 29%, and the average yearly length of hospital stay per patient fell from 83 days to 18 days. In terms of cost-effectiveness, halfway house benefits saved the insurers 59% of their hospitalization costs" (53, p. 1119). Halfway house plus weekly therapy visit equaled 41% of the cost of psychiatric hospitalization benefit alone (53). Several studies have shown day treatment to be cost effective by delaying or avoiding relapse, decreasing symptoms, and changing patients' attitudes toward their illnesses and treatments. Such approaches decrease costs by avoiding hospitalization. Outcomes have been equal to, and sometimes better than, inpatient care (6).

The existing health care system has moved cautiously in this direction. Medicare has covered partial hospitalization since 1987, and Medicaid includes it as a benefit in some treatment settings. Although managed care approaches that emphasize management of high-cost cases allow the provision of extra-contractual benefits on a case-by-case basis for individuals with severe and persistent illness, most private insurance policies do not provide these benefits. As a consequence of the gross deficiency of appropriate incentives for use of a continuum of care for the mentally ill, cost-efficient and effective alternatives such as day treatment are underfinanced and underutilized (54).

The addition of outpatient and partial hospitalization benefits would provide incentives to use those settings more fully. Whatever proposal for universal health insurance is enacted, such incentives are critical if we are to optimally use dollars available for mental illness treatment for the most people. More efficient resource use should increase the appeal of these incentives to payers and policy makers, and because these settings permit more time at home, they are often preferable to patients and their families.

Recognize the Distinction Between Psychotherapy and Medical Management

The diversity of ambulatory psychiatric practice has generally been unrecognized, with payers assuming that all outpatient mental health care was long-term psychotherapy (48). However, beneficiaries also need access to outpatient diagnostic evaluation and acute, medically oriented management. A universal health insurance system that recognizes these treatment needs will allow for early intervention, secondary prevention (i.e., early intervention in relapse), and tertiary prevention (i.e., preventing relapse).

In 1984, the Department of Health and Human Services lifted limits on coverage for Alzheimer's disease (except psychotherapy), so that service type, not diagnosis, determined coverage. This modification recognized different types of outpatient care and established a precedent for further legislative changes (45). As noted earlier, a 1987 legislative modification removed limits from "medical management of psychopharmacologic agents" for Medicare, instituting 20% copayment, at parity with other medical services (45).

Although removing the restrictions for the medical management of mental disorders improves reimbursement, the rules defining medical management must be carefully monitored to ensure that they do not limit reimbursement to brief office visits only. Regulatory wording is critical: a narrow definition risks oversimplification of the concept, which could lead to problems in policy and practice (45). For example, if regulations required that a prescription be written for an office visit to qualify as "medical management," providers might be tempted to game the system.

CURRENT APPROACHES TO UNIVERSAL HEALTH CARE

Legislative activity at the state level has already produced universal coverage in some areas of the country. Hawaii, for example, has had a universal system in place for several years. Oregon's Basic Health Services Act of 1989 guarantees universal access to a basic level of health care (55). A growing number of states have legislative proposals for universal health insurance or minimum benefit plans (56).

The proposals that provide the basis for current debate derive from several sources. Numerous proposals

for universal health insurance have been introduced in the U.S. Congress (some proposals are no longer legislatively active but are nevertheless worthy of examination). In addition, the May 15, 1991, issue of *JAMA* was a forum for the subject of the uninsured. Proposals to solve the access problem were solicited for that issue, and a selection of the best were published. They represented solutions proffered by both politicians and private groups. Table 1 summarizes major proposals from these sources. If they are studied as prototypes, commonalities in their approach to psychiatric care coverage can be elicited. They can also be evaluated for adherence to the principles described earlier.

When one compares benefits in these proposals to the benefits in existing U.S. health insurance programs, their coverage of mental disorders and substance abuse falls into several categories. Benefits in the *MediPlan Act*, the *U.S. Health Act*, the *Davis Proposal*, the *Health Insurance Coverage and Cost Containment Act of 1991*, and the *Health America Plan* resemble or are modeled on Medicare benefits. Several proposals use or resemble private insurance plans, including the proposals modeled on the *Pepper Commission recommendations*, *Health Access America*, *Consumer Choice Health Plan*, and the *Heritage Foundation Proposal*. The *Physicians For a National Health Program*, the *Universal Health Care Act*, and the *Stark Bill* most closely approximate national health insurance systems. A number of the proposals are basic benefit plans, including the *Sabo Bill*, *Durenberger Bill*, *Chandler Bill*, and the *Physicians Who Care Plan*. Several plans combine features of several categories. Of note, none of the proposals to date *specify* mental illness treatment benefits are "nondiscriminatory." All have some type of limit on care for mental disorders (although for those based on Medicare, the limit is primarily a higher copayment for psychotherapy).

All of the national health insurance proposals include hospitalization day limits, which some patients will exceed. For example, 17,000 Medicare beneficiaries have reached their lifetime hospitalization limits (57). With day limits on inpatient psychiatric care, some patients are forced into state hospitals or other forms of uncompensated care. This is yet another reason to preserve and improve the state hospital system.

Limits on all forms of psychotherapy are present in every current national health insurance proposal, usually in the form of visit limits. However, the *Kennedy-Mitchell Bill* and those based on Medicare psychiatric benefits at least recognize the difference between outpatient psychotherapy and medical management of mental illness. The model mental health benefit reimburses psychiatric medical management at parity with medical management of other conditions.

Incentives for a continuum of care are another important principle of benefit design. Of the published versions of current insurance reform proposals, only the *Kennedy-Mitchell Bill* explicitly recognizes the potential for cost-effective use of such alternatives.

The model mental health benefit would ensure the

risk of acute catastrophic expenditures, cover partial hospitalization and residential treatment in a continuum of care, and recognize the distinction between medical management and psychotherapy. The model mental health benefit provides an arena to discuss the nondiscriminatory approaches delineated in this paper. The principles enunciated in this paper are critical if psychiatric patients are to be provided an opportunity for access to quality care.

It is clear that all current proposals fail to some degree the principles elucidated here. Alternative proposals, such as a managed care system with a target of 10% of overall expenditures for mental illness or substance abuse, could also be considered.

RESEARCH AGENDA

Numerous studies are needed to answer the questions put to the profession from within and without. More than ever, the current policy environment necessitates an increase in the overall investment for research on mental illnesses and their treatments. Managed care affects treatment by reducing length of stay, the number of visits for certain conditions, the location of care, and numerous other factors. Financing reform will further affect these parameters. Given these various cost-containment approaches which create incentives for reducing care, the optimal level of care must be determined. Whether the level of care provided now is efficient, is excessive, or constitutes undertreatment is simply unknown (48). Information from outcome studies is needed now more than ever. If treatment protocols with proven utility are developed and implemented, micromanagement by utilization reviewers—one of the more aversive aspects of the current cost-containment process—would be rendered obsolete.

Information requirements include cost-effectiveness and cost-benefit analyses. Ongoing collection of epidemiologic data is needed, as are cost-offset studies and research aimed at determining the optimal use of resources and facilities. Psychiatry has a need for technology assessment as well as for interventions including medications, psychotherapy, psychoanalysis, ECT, and rehabilitation programs. For example, Reifman and Wyatt conservatively estimated that lithium treatment saves \$4 billion annually in savings in treatment and gains from patient productivity. This study supports the hypothesis that improvements in psychiatric care also provide general benefits to society (58).

Similar studies are long overdue. They have intrinsic value in their potential to advance the profession and improve clinical care regardless of policy decisions (58). They are needed whether or not insurance coverage is reformed in order to measure provider performance and highlight areas that need improvement. These studies also help overcome discrimination by payers who believe that all psychiatric illnesses are incurable and a drain on resources (59).

CONCLUSIONS

Even universal health insurance with complete coverage would not be a panacea (60). Mechanic notes that increased insurance coverage is "only one element of a coherent mental health policy" (61, p. 484). Furthermore, all of the current proposals leave patients exposed to financial risks should they reach coverage limits.

If a purely medical approach is too narrow, what should the scope of a reformed system include? Returning to Astrachan and associates' tasks (44), a national health insurance system could improve coverage for the treatment of mental illnesses by extending hospitalization coverage and eliminating discriminatory limits on outpatient visits. It could improve the treatment of the chronically ill and those with rehabilitation needs by covering these services. The public network of rehabilitation programs should be preserved as well. A national health insurance program should not preclude coverage for identified groups such as those remanded to treatment by the legal system and others who society determines need modification or management of their behavior, to be provided by the mental health sector. Coverage of humanistic tasks by universal health insurance is more complex because persons in this category are not always sufficiently functionally impaired to justify expenditure of public or insurance benefits for these services (44). Since long-term care is unlikely to be covered under any form of universal health insurance, continuity of care in the continuum of a service system will require other sources of funding.

The need will remain under any future universal health insurance system for the preservation of a special and justified role for psychiatry. The psychiatric community can advocate a strong public health perspective, encouraging primary prevention of mental illness and substance abuse. Optimal political treatment of these obligations will be more likely if the psychiatric profession is unified in its opinion about the relative importance and practicality of funding each of these tasks in any reformed system.

Patterning psychiatric benefits in a national or universal health insurance program after the model mental health benefit would be a start toward these goals, by basing psychiatric coverage on nondiscriminatory insurance principles that encourage parity with other conditions and minimize risk of catastrophic expenses. If universal health insurance is to be comprehensive and promote access, more resources will be needed to reduce patient cost sharing (i.e., eliminate the 1-day deductible and the 50% copayment) and to enrich long-term care and adjunctive social services.

If the basic benefit approach is to be adopted, then limits can be imposed on inpatient stays and psychotherapy visits, with the understanding that this will lead to increased use of the public sector or bad-debt risk pools. That is the "price" of favoring access over catastrophic coverage: everyone gets a little, while some are still left unprotected or relegated to second-class care.

Reform decisions will be political, but psychiatrists' input to the process is needed.

We have attempted to delineate principles with regard to psychiatric care that can be used to evaluate the merits of any universal health insurance proposal. There is reason for optimism regarding our potential for accomplishment in future insurance reform, for while no current proposal covers treatment as it should under these principles, features of some proposals approach them. The issue deserves close scrutiny by all providers.

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The Phenomenological and Conceptual Interface Between Borderline Personality Disorder and PTSD

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***Objective:** The authors explore the conceptual and phenomenological interface between posttraumatic stress disorder (PTSD) and borderline personality disorder as well as the therapeutic and research implications of this interface. **Method:** They systematically review the relevant empirical, conceptual, and clinical literature. **Results:** These seemingly separate disorders are related. Borderline personality disorder is often shaped in part by trauma, and individuals with borderline disorder are therefore vulnerable to developing PTSD. **Conclusions:** The authors draw a distinction between the enduring effects that traumas can have on formation (or change) of axis II personality traits (including those found in borderline personality disorder) and acute symptomatic reactions to trauma, called PTSD, that are accompanied by specific psychophysiological correlates. They describe the implications of these conclusions for DSM-IV, therapy, and future research.*

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This examination of the interface between borderline personality disorder and posttraumatic stress disorder (PTSD) should be placed in the perspective of examinations of two other axis I disorders with which borderline psychopathology has been linked. Previous examinations of the interface between borderline personality disorder and schizophrenia (1, 2) and affective disorder (3) have involved research that helped establish the present boundaries for the borderline construct but have not greatly enhanced understanding of the pathogenesis of this personality disorder. Our current review of the relationship between PTSD and borderline personality disorder indicates that viewing the link between borderline personality disorder and childhood trauma through the lens provided by PTSD can cast more substantial light on the pathogenesis of borderline personality disorder.

Borderline personality disorder and PTSD both became official diagnoses in 1980 with the appearance of DSM-III. Awareness of posttraumatic psychiatric consequences has been present since the Civil War, but it was the influx of casualties from the Vietnam conflict that led to its inclusion in our nosology. As a symptom complex with physiological correlates, triggered by an external event, PTSD entered the axis I section of DSM-III. The earlier literature on PTSD was largely derived

from the study of combat veterans and included a variety of approaches to its treatment (4-7). In contrast, borderline personality disorder entered the axis II section of DSM-III as a type of personality organization. Its inclusion in DSM-III grew out of its already widespread use by psychoanalytic clinicians and the empirically based descriptive studies that clinical use of the concept had already prompted (8).

The emergence of both borderline personality disorder and PTSD as official diagnostic categories has been greeted by wide but not always discriminating use. For many clinicians a diagnosis of "borderline" remains synonymous with "severe" personality disorder (9, 10), which is akin to Kernberg's original use of the term (11). According to Vaillant (12), the diagnosis is used for any patient who evokes hostile countertransference reactions. In this respect, feminist clinicians have voiced the concern that overuse of the diagnosis of borderline personality disorder by male clinicians for female patients reflects a negative attitude toward these patients (13). When Morey and Ochoa (14) examined this concern empirically, they found it to be unjustified, but the idea that the diagnosis of borderline personality disorder is pejorative has doubtless fueled enthusiasm for using diagnoses that evoke more supportive attitudes—such as PTSD.

The use of PTSD has been encouraged by the fact that it is a relatively efficient way to make a diagnosis that is both discrete and understandable to patients and to third-party payers. Moreover, public awareness of child abuse and the current mental health climate has made it professionally shameful to overlook or minimize a history of abuse. Unfortunately, hurried clinical

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TABLE 1. Studies of Childhood Trauma

Authors	Year	N ^a	Diagnoses of Comparison Subjects	Childhood Experiences of Subjects With Borderline Personality Disorder (%)			
				Separation/ Loss	Physical Abuse	Sexual Abuse	No Trauma
Soloff and Millward (17)	1983	45	Schizophrenia, depression	56–62 ^b			
Akiskal et al. (18)	1985	100	Other personality disorder, bipolar disorder, depression	37 ^b			
Links et al. (19)	1988	88	Subthreshold borderline criteria	25 ^b	29 ^b	26 ^b	44 ^b
Zanarini et al. (20)	1989	50	Other personality disorder, antisocial personality disorder	46 ^b	46	26 ^b	26
Herman et al. (21)	1989	24	Other personality disorder		71 ^b	67 ^b	19 ^b
Johnson et al. (22)	1989	43	Other personality disorder, normal	40			
Ogata et al. (23)	1990	24	Depression		42	71 ^b	21 ^b
Shearer et al. (24)	1990	40			25	40	
Stone (25, 26)							
New York Hospital-Westchester Division study	1987	29			28	35	
New York Psychiatric Institute study	1990	206	Schizophrenia, schizoaffective disorder	37	11	17	45
Australian study	1987	15				47	
Westen et al. (27)	1990	23	Undefined		52	52 ^b	
Paris and Zweig-Frank (28)	1992	78	Other personality disorder		70 ^b	70 ^b	27 ^b

^aNumber of subjects with borderline personality disorder.

^bSignificantly higher than percent of comparison patients.

cians may too readily diagnose anyone who has had recent exposure to severe stress as having PTSD—despite the fact that fewer than a fourth of patients who experience such stress actually develop the axis I syndrome (15).

Although borderline personality disorder and PTSD had very different backgrounds before entering DSM-III, their interface was propelled into attention by the overlap in their DSM-III and DSM-III-R definitions and acquired great significance after Herman and van der Kolk (16) highlighted the role of childhood trauma in the formation of borderline psychopathology. Several studies have persuasively documented that many patients with borderline personality disorder have childhood histories of sexual and/or physical abuse (17–28) (table 1). Available epidemiologic data indicate that of the 2%–3% of the population with borderline personality disorder (29, 30), roughly one-third fulfill criteria for PTSD (29). The overlap between the phenomenology of borderline personality disorder and the phenomenology of PTSD raised the same questions about their relationship as had been previously raised about the relationship between borderline personality disorder and schizophrenia and affective disorder. Are they separate but comorbid conditions? Does one contribute to the development of the other? Do they share common etiological backgrounds to the point that they are variants of the same disorder, and, if so, is this overlap in etiology sufficient that the disorders should be classified together on either axis II or axis I?

In this paper we will review the phenomenological and conceptual interface between borderline personal-

ity disorder and PTSD and document that these seemingly separate diagnoses have a complicated relationship. We will also identify areas where available knowledge can lead clinicians to more discriminating use of these diagnoses and, perhaps, eventually lead to more effective treatment strategies. In this process, we will identify multiple areas that are in need of more study.

THE PHENOMENOLOGICAL INTERFACE: SYMPTOMS AND TRAITS

In 1987, Herman and van der Kolk (16) observed that the clinical presentation of patients with either PTSD or borderline personality disorder shared major disturbances in affect regulation, impulse control, reality testing, interpersonal relationships, and self-integration. We will show that their overlap in presentation can often mask distinctions found by taking careful longitudinal and developmental histories.

Many characteristics of PTSD, if enduring, could also represent traits that offer evidence for a disorder of personality. This includes four of the proposed DSM-IV avoidant criteria (C4–C7) and two of the arousal criteria (D2 and D4) for PTSD (31) (appendix 1). If these or other symptoms of PTSD emerge and persist after exposure to a severe stressor, they represent a personality change (not necessarily like borderline personality disorder) for which DSM-IV now offers a new category—personality change after catastrophic experience (32). If several of these symptoms, namely hypervigilance (D4), irritability (D2), and feeling detached or es-

TABLE 2: Perceptual Problems Reported in Studies of Patients With Borderline Personality Disorder

Authors	Year	N ^a	Visual Illusions	Reported Perceptual Problem					
				Dissociation			Paranoid Experience		
				Deperson-alization	Dereal-ization	Undefined	Ideation	Ideas of Reference	Undefined
George and Soloff (37) ^b	1986	30	33		37				
McGlashan (38) ^b	1987	81	12	9	9		11	9	
Widiger et al. (39) ^b	1987	27	21				12	25	
Frances et al. (40)	1984	26				30			
Chopra and Beatson (41)	1986	13		85	92				77
Jacobsberg et al. (42)	1986	22	36	73			73	77	
Links et al. (43)	1989	88		49					32
Silk et al. (44)	1990	24				37			37
Zanarini et al. (45)	1990	50	24	36	30		78	74	

^aNumber of subjects with borderline personality disorder.

^bPatients with borderline personality disorder with comorbid schizotypal personality disorder were not included in study.

tranged (C5), were to emerge, endure, *and* be associated with an intense unstable relationship or self-destructiveness, the diagnosis of borderline personality disorder might wrongly be made. This mistake would reflect a failure to attend to the early onset requirement for a personality disorder diagnosis.

The overlap between PTSD and borderline personality disorder is further confounded by the fact that instability is central to the construct of borderline personality disorder and a number of its criteria. If certain traits (namely, borderline criteria 5, 6, and 8 and the newly proposed criterion 9 [appendix 1]) are not documented parts of enduring patterns, they could be considered symptoms rather than personality traits. Their use as criteria for a personality disorder rests on their having an early onset and their being persistent qualities that endure across many situations. Moreover, other criteria for borderline personality disorder (e.g., criteria 1 and 3), intended to describe ongoing stable traits, could also surface as symptomatic reactions to trauma and—if their durability is unappreciated—could then wrongly be seen as evidence for a disorder like PTSD, if not PTSD itself.

Although the phenomenology of borderline personality disorder and PTSD would seem to be very intertwined, clinicians can usually distinguish these disorders by applying a fundamental distinction between symptoms and traits. The prototypical patient with PTSD presents with a subjectively disturbing symptom complex occurring in reaction to exposure to a recognizable and extreme stress (combat, rape, assault, etc.). PTSD usually is not confused with borderline personality disorder if the patient has a history of reasonably healthy functioning and relationships. The prototypical patient with borderline personality disorder presents with a hunger for care and a history of repeatedly failed relationships that would be atypical for most patients with PTSD. Thus, disentangling the overlap in phenomenology involves taking a careful longitudinal history to distinguish the enduring patterns that constitute personality traits from the rapid, adult-onset, symptomatic occurrence of those phenomena which overlap.

Although a longitudinal perspective can often distinguish when phenomena are symptoms of PTSD or traits of borderline personality disorder, unusual perceptual phenomena such as dissociation, paranoid ideation, or visual illusions may offer a new overlap issue in DSM-IV. Both dissociative and paranoid experiences have been associated with borderline personality disorder since before DSM-III (33, 34) and well before its linkage to PTSD. These symptoms are understood as observable evidence of the vulnerable reality sense and reality testing that characterize borderline psychopathology (11, 35, 36). Indeed, the prevalence and discriminating power of these experiences (table 2) has led to their being proposed as a new ninth criterion for borderline personality disorder in DSM-IV (appendix 1) (9). Dissociative experiences might also be either a reexperiencing (B3) or avoidant (C5) criterion for PTSD (appendix 1), and paranoid experiences are identifiable in several PTSD criteria such as B4, D2, and D4 (appendix 1). For borderline patients such experiences are usually triggered by an interpersonal stimulus (46), such as the perception of being abandoned or rejected, an experience believed to reflect intolerance of being alone (35). The dissociative states of borderline personality disorder often involve depersonalization and derealization. Unlike such states in PTSD, the dissociative states of borderline personality disorder are not commonly associated with amnesia, fugue, or problems with concentration. Moreover, the borderline patient's dissociative episodes are usually more transient and milder than those of the patient with PTSD, which, in DSM-III-R, requires a month's duration. DSM-IV proposes that the more transient types of dissociative states found in borderline personality disorder should be distinguished from PTSD as a separate category—brief reactive dissociative disorder (31).

The most distinctive signals for the presence of PTSD are the reexperiencing symptoms—intrusive memories, recurrent nightmares, or flashbacks (criteria B1–B3). Their occurrence reflects sensitivity to trigger stimuli (internal or external cues, criterion B4), such as smell, body position, or window lighting, that symbolize or

resemble an aspect of the traumatic experience. The presence of these aspects of PTSD is closely related to the severity of the stressor event and not closely related to premorbid vulnerability (47). It is our impression that although these reexperiencing symptoms are not typical of most borderline patients, the types of visual illusions experienced by some borderline patients (table 2) often appear after exposure to current stresses and—now that the connection between borderline personality disorder and trauma has been established—may overlap the flashbacks or illusions of reliving of PTSD (criterion B3). When reexperiencing symptoms occur in borderline patients, DSM-III-R considers their appearance to be evidence for a comorbid problem, namely, PTSD. The emergence of PTSD symptoms under such circumstances could also be considered an elaboration of borderline personality traits (e.g., mood reactivity, abandonment fears, and vulnerable reality testing) caused by the strains placed on usual defenses. In any event, vulnerability to such reexperiencing symptoms (and comorbid PTSD) will probably be found to join other features (i.e., promiscuity, dependent/masochistic relationships, chronic dysphoria, and dissociative experiences) that have been shown to distinguish patients with borderline personality disorder who have histories of childhood abuse from those who do not (23, 26).

There is considerable phenomenological overlap in the ways that both PTSD and borderline personality disorder can present, such as being desperate, impulsive, self-destructive, and angry. There are also fundamental distinctions between the traits belonging to borderline personality disorder and symptoms belonging to PTSD that can be identified by use of a longitudinal perspective. Although perceptual symptoms can occur with both disorders, these may be more transitory and relationship-related for borderline personality disorder and more directly associated with traumatic memories for patients with PTSD. This is an emerging area that still requires more empirical examination. Other empirically testable questions include the following: 1) Can the phenomenology of borderline psychopathology be divided into parts that do and do not represent posttraumatic consequences? 2) Do different types of childhood trauma (e.g., abandonment versus abuse) or do the ages, frequency, or relationship to the perpetrator have distinctive consequences in the phenomenology of patients with borderline personality disorder?

THE CONCEPTUAL INTERFACE: TRAUMA AND PERSONALITY

Borderline personality disorder has become part of the still-enlarging awareness of the high frequency and manifold effects of trauma. This linkage can be traced retrospectively to Adolph Stern's seminal 1938 paper on borderline psychopathology (48). He noted that "actual cruelty, neglect and brutality by the parents of many years' duration are factors found in these patients" (48). Although a few other clinicians subse-

quently noted a high frequency of incest (49, 50), most psychoanalytically oriented writers overlooked Stern's observation. Even before Herman and van der Kolk observed the overlap in the phenomenology of borderline personality disorder and PTSD, Browne and Finkelhor (51) reported that victims of sexual abuse develop a constellation of symptoms that clinicians could readily associate with borderline personality disorder—specifically, depression, substance abuse, revictimization, and self-destructiveness. Subsequently, the link of childhood abuse to self-destructiveness—the most prototypical borderline behavior (14, 35)—has been confirmed (52), and a link with dissociation has also been demonstrated (53). This work has set the stage for the examination of how such childhood experiences relate to developing borderline personality disorder.

Available epidemiologic evidence suggests that although 38% of the population is exposed to catastrophic stress, only about 9.2% ever experience a PTSD-like reaction (15). Hence, the PTSD construct is complicated by the issue of vulnerability. It has been observed that PTSD is more likely to occur in individuals who have had previous exposure to unusual stress (54) or who have maladaptive coping mechanisms (55) and other adjustment problems (56). Recently Breslau et al. (15) identified childhood separations, a family history of antisocial behavior, and being female as risk factors for developing PTSD. The majority (75%) of patients with borderline personality disorder are women (9), and many have had excessive exposure to childhood separation experiences (table 1); patients with borderline disorder also have a high rate of antisocial behavior in their first-degree relatives (57). Therefore, they are also vulnerable to PTSD. This vulnerability is verified by the fact that roughly a third of patients with borderline personality disorder meet DSM-III-R criteria for PTSD (29).

The abusive experiences of borderline patients occur significantly earlier—mainly in childhood or during latency (20, 21, 27)—and more often involve the child's caregivers (23, 24, 27, 28) than do the abusive experiences of other clinical populations. These characteristics of the trauma underscore their severity and the likelihood that they will exert enduring effects on the child's character. In response to such trauma, children can become emotionally detached, forget, and dissociate; children can even develop PTSD (58). Beyond such observable reactions, the traumas evoke immature or primitive defenses that can persist well after the stress is over (59). It seems likely that the image-distorting defenses such as denial, splitting, and projection found to typify borderline patients (11, 60, 61) are at least in part derivative of the childhood traumas that most borderline patients have experienced. Thus, a dialectical process occurs after trauma whereby stress causes poor coping mechanisms that predispose to more stress and to having more maladaptive reactions to such stress. Given that premorbid vulnerability plays a relatively small role in the development of PTSD in reaction to extreme stress (5, 62, 63), having borderline person-

ality disorder doubtlessly represents such extreme vulnerability that even modest stress may evoke it.

In the language of object relations, trauma affects representations of the self (e.g., as bad or helpless) and of others (e.g., as punitive or powerful) that shape the child's future expectations, goals, and reactions. More globally, the traumatized child's type of attachments are characterized by approach-avoidance and "hyper-alert watchfulness" (64, 65). These concepts are core aspects of the borderline personality disorder construct, and the terms are consistent with an axis II perspective.

In neurophysiological language, the model of "kindling" can be used to explicate this process (66). According to this model, childhood traumas leave lasting traces in the brain's neurophysiological regulatory system; these are subsequently represented by hypersensitivity to later stressors that are evocative of the original trauma—stressors that would otherwise not reach the threshold for eliciting the excitation observed (67, 68). These are also core aspects of the PTSD construct, and the terms are more consistent with an axis I perspective.

Although abnormal neurophysiology of serotonin has been proposed as a temperamental substrate for borderline personality disorder psychopathology to account for hostility and impulsivity (69), this abnormality could as readily be the "carrier" condition that results from early trauma. Patients with PTSD show greater sympathetic activity, hyperfunction of the hypothalamic-pituitary axis, and dysregulation of the endogenous opioid system (70). These neurophysiological "carriers" may be important determinants of whether disturbances of personality develop after later exposure to traumas (71). In fact, Kolb (72) postulated that neuronal change in PTSD leads to inescapable physiological disturbances that in turn distort body image, self-concept, and, ultimately, personality.

Because several studies (table 1) have found a high frequency of childhood trauma in subjects who develop borderline personality disorder, because family studies (25, 49, 73) have shown that borderline personality disorder develops without abuse or abandonment about 20%–40% of the time, and because a history of childhood abuse is also associated with many other types of psychopathology (74, 75), it seems safe to conclude that the role of abuse in the pathogenesis of borderline psychopathology, although important, is neither specific nor sufficient. Borderline psychopathology arises out of a history in which abusive experiences join other factors to help shape enduring aspects of the character. The abuse in such instances is symptomatic of more pervasive problems of enduring emotional neglect and extreme conflict with both parents (20, 19, 25, 73, 76). The impact of abusive experiences, like the impact of experiences of loss (77), is probably mediated by the exposure to a neglectful environment before and after the events. Supportive caregiving can greatly diminish the consequences of exposure to abuse and, presumably, other trauma and can result in healthy adaptation to the experience (78).

The clinical diagnosis of borderline personality disorder

is apt for describing a personality whose warp and woof can be traced to a complex intermingling of familial emotional neglect and misunderstanding beginning in childhood—often amplified by and perhaps partly created by the occurrence of childhood traumas (21, 57, 79).

These comments highlight a broader conceptual issue. Psychoanalytic theory holds that personality is formed, to a great extent, from the interaction between the child's innate disposition (i.e., his or her constitution or temperament), early parenting experiences, and other childhood experiences. Early psychoanalytic theory sees personality developing as "compromise formations" from the conflicts between impulses and prohibitions. These compromise formations organize the way a child binds and channels energy and organize the child's experiences and expectations. The emergence of object relations theory has added weight to the issue of whether such prohibitions are offered by the primary caregiver and within a predictable empathic relationship. In any event, personality development is intimately connected to the developmental processes involving stress, restraint, and frustration, and what renders these developmental experiences traumatic as opposed to creative depends a great deal on whether, in retrospect, we believe the resultant character traits are adaptive or not. Beyond the more easily identifiable traumas of physical or sexual abuse, desertion, and abandonment are the less obvious "microtraumas" such as emotional neglect, humiliation, or misattribution of blame (80). The boundary between the situational events and interpersonal problems that are identifiable as traumatic and those which are identifiable as creative is intrinsically unclear and subtle. Those children who experience severe traumas, with or without developing childhood PTSD, will adapt in ways that represent compromise formations which allow them to creatively adapt and survive. Once created, these compromise formations represent consolidated coping patterns that, unfortunately, may subsequently prove maladaptive in other, nontraumatic contexts. The image-distorting defenses used by borderline patients exemplify such handicaps.

CONCLUSIONS

We have described how the axis I diagnosis of PTSD was developed to identify a syndrome occurring in adults as a result of exposure to extreme stress. It was not intended to describe a childhood-initiated developmental process. Predisposing factors in personality (immature defenses or coping mechanisms), a family history of antisocial behavior, early separation experiences, and exposure to previous sustained traumas all make people with borderline personality disorder more likely to develop PTSD in response to later stress. Borderline personality disorder exemplifies a type of personality whose formation is often partly shaped by childhood traumas—a type of personality that is keenly vulnerable to developing PTSD in response to what

might for others be subthreshold stressors. Having noted this, we think that these findings have implications for our diagnostic system, for treatment, and for future research.

The interface between borderline personality disorder and PTSD has several implications for DSM-IV. First, the proposed changes in the criteria for borderline personality disorder (defining persistent self-image distortions in criterion 3 and, as discussed, adding a ninth criterion reflecting cognitive/perceptual disturbance) highlight phenomena with associations to childhood trauma. We believe that this will usefully anchor the phenomenology of this personality disorder in its pathogenesis—as well as giving recognition to clinically important features (9). Second, DSM-IV should better convey the reciprocal relationship between severity of the stressor and premorbid vulnerability in developing PTSD. The emergence of PTSD symptoms after exposure to only a mild stressor should alert clinicians to the need to clarify vulnerability issues, and borderline personality disorder should specifically be cited as a known form of vulnerability. Moreover, the DSM-IV description of borderline personality disorder should cite PTSD as a common comorbid and complicating condition, as proposed by Davidson and Foa (81). Third, emphasis should be given to the distinctions between axis II traits of borderline personality disorder and axis I symptoms of PTSD by urging attention to their onset and duration. Fourth, added weight should be given to the reexperiencing subset of PTSD criteria in view of their relative independence from vulnerability. Clinicians should be urged to distinguish when the avoidant or arousal criteria of PTSD become enduring traits as opposed to reactive symptoms. Fifth, when PTSD symptoms become enduring traits as a result of adult stress, the reaction should be identified—regardless of how borderline personality disorder-like the illness seems—as personality change after catastrophic experience (32).

The enhanced understanding of the interface between borderline personality disorder and PTSD also has implications for treatment. Clinicians should be aware that their countertransference reactions may be operative when they use either of these diagnoses. Misuse of either PTSD or borderline personality disorder may reflect sympathy or dislike, respectively, as countertransference problems. The hazards associated with dislike of a patient with borderline personality disorder may have impelled the current enthusiasm for sympathy for the patient with PTSD.

Acknowledgment of the past traumas of borderline patients, accompanied by appreciation for the adaptive value of the ways in which the person managed the experience as either a child or an adult, can help the borderline patient attach to a therapist and ally with therapeutic goals (61, 79). Because recognition of a history of childhood trauma encourages a more supportive attitude, this can diminish the frequency with which borderline patients drop out of psychotherapy due to negative reactions to early interpretations or confrontations

(82, 83). It can also lead to a more cautious and discriminating approach to family involvement (79).

Recognition of PTSD as a comorbid condition for borderline patients (i.e., those whose traumas are evoked and being reexperienced) should encourage the use of support groups. Such groups have proven effective in diminishing the pathological impact of trauma (5, 80). This use of groups contrasts with the usual unresponsiveness to group therapy referrals that has characterized most patients with borderline personality disorder (35, 84). Patients with borderline personality disorder and comorbid PTSD might also respond to medications directed at the reexperiencing symptoms such as propranolol (85, 86) or phenelzine (87). Conversely, recognition of comorbid borderline personality disorder may help explain variations in the response to medications among patients with PTSD (88).

Beyond these ways in which the recognition of trauma can inform treatment of borderline personality disorder, it is also important in later stages of treatment that a focus on traumas not deflect attention from the characterologic vulnerability issues (e.g., aggressive and secondary gain motives) that often complicate dysfunctional behaviors. This process differentiates here-and-now from past experiences of neglect or abuse and is needed in order to help modify the borderline patient's maladaptive character structure.

We have already identified a number of empirical questions that still surround the interface between borderline personality disorder and PTSD. First, could borderline psychopathology develop from repeated childhood exposure to abuse in the context of a reasonably healthy family? Second, do childhood histories of sustained neglect and periodic trauma predictably lead to developing borderline personality disorder? Third, can the neurophysiological changes associated with the PTSD syndrome also be found as sequelae to childhood trauma in the neurophysiological substrate of borderline personality disorder? If so, can these changes be altered pharmacologically in ways that diminish the character-forming consequences of trauma in children or the duration of the PTSD symptoms when they emerge in borderline adults? Finally, do borderline patients with histories of severe/sustained abuse differ significantly in their prognosis and/or treatment response from those without such histories? If they do, does this signify that the borderline construct should be narrowed to exclude patients whose character did not develop out of such trauma? If this should happen, borderline personality disorder would become a prototype for a personality disorder with a description strongly anchored in the medical tradition of being etiologically based and therapeutically specific. It might then assume a place alongside yet another axis I condition, multiple personality disorder.

In this paper we have examined the relationship between an axis I disorder, PTSD, and an axis II disorder, borderline personality disorder. In the process, the limitations of such a division and the complexities of the interface between them have been uncovered. Unlike

the previous examinations of the interface between borderline personality disorder and other axis I disorders, we found that trauma offers an important perspective on its pathogenesis. As with other axis I/axis II divisions, the presence of seemingly separate but related categories in our diagnostic system provides a structure and a stimulant for further research. We have identified some directions that can be taken to further illuminate this interface.

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APPENDIX 1. Proposed DSM-IV Criteria for Borderline Personality Disorder and PTSD From *DSM-IV Options Book: Work in Progress 9/1/91* (31)

Criteria for Borderline Personality Disorder

Pervasive pattern of instability of interpersonal relationships, as indicated by five or more of the following:

1. Frantic efforts to avoid real or imagined abandonment
2. Pattern of unstable, intense interpersonal relationships

3. Identity disturbance: persistent self-image distortions (e.g., feeling that one does not exist or embodies evil) (this criterion involves significant changes from DSM-III-R)
4. Impulsiveness in potentially self-damaging areas (e.g., substance abuse)
5. Recurrent self-destructive threats, gestures, or behavior
6. Affective instability; marked reactivity of mood (e.g., intense episodic dysphoria)
7. Chronic feelings of emptiness
8. Inappropriate, intense, or uncontrolled anger
9. Transient, stress-related dissociative or paranoid ideation (this criterion involves significant changes from DSM-III-R)

Criteria for PTSD

- A. Exposure to traumatic event or severe stress
- B. Reexperience of the traumatic event in one or more of the following ways:
 1. Recurrent and intrusive recollections (images, thoughts, perceptions) of the event
 2. Recurrent distressing dreams of the event
 3. Acting or feeling as if event were recurring (flashbacks or illusions of reliving the event)

4. Intense psychological reaction to internal or external cues symbolizing or resembling the event
5. Physiological reactivity to internal or external cues symbolizing or resembling the event
- C. Persistent avoidance of stimuli associated with the event, indicated by two or more of the following:
 1. Avoidance of thoughts and feelings associated with the trauma
 2. Avoidance of activities associated with the trauma
 3. Inability to recall the trauma
 4. Diminished interest in significant activities
 5. Feeling detached or estranged
 6. Restricted affect (e.g., unable to have loving feelings)
 7. Sense of a limited future
- D. Persistent arousal symptoms, indicated by two or more of the following:
 1. Difficulty falling or staying asleep
 2. Irritability
 3. Difficulty concentrating
 4. Hypervigilance
 5. Exaggerated startle response
- E. Duration (symptoms for 1 month or more or 3 months or more or subspecify as acute or chronic) (these criteria involve significant changes from DSM-III-R)

Network Therapy for Addiction: A Model for Office Practice

Marc Galanter, M.D.

Individual therapists in office practice are often considered to have limited effectiveness in treating alcohol and drug dependence. In this article the author describes network therapy, an approach developed to assure greater success in such treatment. It uses psychodynamic and behavioral therapy while engaging the patient in a support network composed of family members and peers. A cognitive-behavioral model of addiction, based on the role of conditioned withdrawal in relapse, is described. Related techniques for securing abstinence are then reviewed; they augment individual psychotherapy to help patients avoid relapse caused by the affective and environmental cues that precipitate drug seeking. The role of social cohesiveness as a vehicle for engaging patients in treatment is outlined next, along with a related technique for enhancing an addicted patient's commitment to the therapy. This is done by using the patient's family and peers as a therapeutic network to join the patient at intervals in therapy sessions. The network is managed by the therapist to provide cohesiveness and support, undermine denial, and promote compliance with treatment. The author presents applications of the network technique designed to sustain abstinence and describes means of stabilizing members' involvement. Applications of network therapy to ambulatory detoxification, disulfiram and naltrexone administration, relapse prevention, and contingency contracting are reviewed.

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Addictive illness is a highly prevalent psychiatric disorder and a well-recognized national priority. Nonetheless, techniques for rehabilitating alcohol- and drug-dependent patients in office practice are not well established (1). Even psychiatric residency programs have only recently been required to provide training for treatment of such patients, and such training often focuses primarily on problems of detoxification (2). The task of developing rehabilitation techniques is therefore important if substance dependence is to be managed within the mainstream of mental health, without routinely turning to specialized inpatient units. To this end, I shall describe a cognitive-behavioral model of addictive behavior relevant to ambulatory care and a related approach to individual therapy that draws on techniques for engaging support from family and peers.

NEED FOR A NEW PERSPECTIVE

Substance abuse treatment is as great a challenge for psychiatry as any clinical issue that has emerged in recent decades. This was evident in the Epidemiologic

Catchment Area Study, where the lifetime prevalence of abuse and dependence across study sites was 15% for alcohol and 6% for other drugs (3). These were the disorders with the highest prevalences for men of all age groups and the highest prevalences for women between ages 18 and 24 years (4). The cost of alcohol and drug problems to American society in health care, lost productivity, and law enforcement is also onerous. It is greater than that of all other mental illnesses combined (\$144 versus \$129 billion in 1985) (5).

Furthermore, support for addiction treatment is not now expanding. Recognition of the severity of the substance abuse problem in the 1970s initially led to an increase in inpatient care. The availability of beds in designated units increased by 62% from 1977 to 1984, and all of this net gain was in the private sector (6). During this time, the "Minnesota model" for inpatient management (7), based on a protracted inpatient stay, became a standard of treatment for many middle-class substance abusers. A recent wave of cost containment, however, has led bed occupancy rates in nonpublic facilities to fall as low as 60% (8); the decline has been fueled by a lack of evidence for the relative advantage of inpatient over ambulatory clinic care (1).

Managing addicted patients in office practice may potentially be less costly, but reports on its relative effectiveness have not been positive. Hayman (9), in an early survey of psychiatrists in practice, found that very few professed an appreciable degree of success in treating alcoholics in office practice. No difference in outcome was found when outpatients were offered individ-

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ual therapy as a treatment added to medical monitoring alone (10), nor was insight-oriented therapy found to enhance the effectiveness of outpatient milieu treatment for alcoholism (11). Indeed, Vaillant (12), commenting on alcoholism treatment, said, "The greatest danger of this is wasteful, painful psychotherapy that bears analogy to someone trying to shoot a fish in a pool. No matter how carefully he aims, the refracted image always renders the shoot wide of its mark." As conventionally practiced, individual therapy does not appear to be an effective tool for addiction rehabilitation.

A number of issues, however, can be considered in formulating a new approach to augment office-based therapy. Recent years have witnessed both research-based and clinically based support for the importance of securing abstinence as an initial step in addiction treatment, rather than awaiting results of an exploratory therapy (13-15). This position has been strengthened by the widespread acceptance of Alcoholics Anonymous (AA), itself strongly oriented toward abstinence. To implement a regimen of abstinence, clinical researchers have developed a number of structured techniques, focusing on cognitive-behavioral change (16, 17) and interpersonal support from family and peers (18-20). In this article I shall consider how these approaches can be adapted to an office practice oriented toward individual therapy so as to secure abstinence and effective rehabilitation.

I call this integrated approach "network therapy" because it draws on the supportive role of a group of family and peers introduced into therapy sessions. The term derives from the work of Speck and Attneave (21), who used a large support group drawn from the patient's family and social network as a tool for psychiatric management. They used these networks for both psychological and practical aid in addressing acute psychiatric illness so as to avert a hospitalization until the acute symptoms remitted. Once mobilized, the network was then available to aid in ambulatory rehabilitation as well. To define what this approach must accomplish, though, it is first necessary to examine some unique characteristics of the substance dependence syndrome.

A BEHAVIORAL MODEL AND AMBULATORY CARE

For many clinicians, the problems of relapse and loss of control, embodied in the first two criteria for substance dependence in DSM-III-R, epitomize the pitfalls inherent in addiction treatment. Because addicted patients are typically under pressure to relapse and ingest alcohol or drugs, they are seen as poor candidates for stable treatment. Loss of control has been used to describe addicts' inability to reliably limit consumption once an initial dose is taken (15).

Conditioned Abstinence

These clinical phenomena are generally described anecdotally but can be explained mechanistically as well

by recourse to the model of conditioned withdrawal, one that relates the pharmacology of dependency-producing drugs to the behaviors they produce. Wikler (22), an early investigator of addiction pharmacology, developed this model to explain the spontaneous appearance of drug craving and relapse. He pointed out that drugs of dependence typically produce compensatory responses in the central nervous system at the same time that their direct pharmacologic effects are felt, and these compensatory effects partly counter the drug's direct action. Thus, when an opiate antagonist is administered to addicts maintained with morphine, latent withdrawal phenomena are unmasked. Similar compensatory effects are observed in alcoholics maintained with alcohol, who evidence evoked response patterns characteristic of withdrawal while still clinically intoxicated (23).

Wikler studied addicts maintained with morphine and then thrown into withdrawal with a narcotic antagonist. After several trials of precipitated withdrawal, he found that a full-blown withdrawal response could be elicited in his subjects when a placebo antagonist was administered. He concluded that the withdrawal had been conditioned and was later elicited by a conditioned cue, in this case the syringe used to administer the placebo. This hypothesized mechanism was later confirmed by O'Brien et al. (24), who elicited conditioned withdrawal by using sound tones as conditioned cues. This conception helps to explain addictive behavior outside the laboratory. A potential addict who has begun to drink or use another drug heavily may be repeatedly exposed to an external stimulus (such as the sight of a liquor bottle) or an internal one (such as a certain mood state) while drinking. Subsequent exposure to these cues may thereby produce conditioned withdrawal symptoms, subjectively experienced as craving. A dramatic example of this phenomenon is often seen among heroin addicts, in whom a severe withdrawal syndrome may emerge when they return to the neighborhoods where they have previously used heroin, even after years away from the drug. In this case, the setting of the neighborhood itself serves as a cue that produces the symptoms and signs of withdrawal (25, 26).

Implications for Treatment

This model helps to explain why relapse is such a frequent and unanticipated aspect of addiction treatment. Exposure to conditioned cues, ones that were repeatedly associated with drug use, can precipitate reflexive drug craving during the course of therapy, and such cue exposure can also initiate a sequence of conditioned behaviors that lead addicts to relapse unwittingly into drug use.

Loss of control can be the product of conditioned withdrawal, described by Ludwig et al. (27) and long recognized on a practical level by members of AA. The sensations associated with the ingestion of an addictive drug, such as the odor of alcohol or the euphoria pro-

duced by opiates, are temporally associated with the pharmacologic elicitation of a compensatory response to that drug and can later produce drug-seeking behavior. For this reason, the "first drink" can serve as a conditioned cue for further drinking. Patients therefore have a very limited capacity to control consumption once a single dose of drug has been taken.

Case 1. A 30-year-old cocaine addict undergoing treatment had been abstinent and well motivated for 2 months but occasionally drank socially. One evening he sought out a cocaine dealer on an impulse and purchased and then insufflated 1 g of cocaine. After returning home, he bought more cocaine and continued to take the drug over the course of the entire night. Examination of this sequence of events in his next therapy session revealed that he had been sitting in a restaurant bar that evening with a date whom he knew to use cocaine. After having two drinks, he had gone to the restroom, where he had used cocaine at times before entering treatment. It was after this that he bought the cocaine. The patient acknowledged that he had simultaneously been exposed to a number of cues which had regularly been associated with his previous cocaine consumption: a sexually charged situation with a cocaine user, alcohol consumption, and a physical setting where cocaine had been frequently used.

Changes in mood state can also become conditioned stimuli for drug seeking, and the substance abuser can become vulnerable to relapse through reflexive response to a specific affective state. Such phenomena have been described clinically by Khantzian (28) as self-medication. Such mood-related cues, however, are not necessarily mentioned spontaneously by the patient in a conventional therapy. This is because the triggering feeling may not be associated with a memorable event, and the drug use may avert emergence of memorable distress.

More dramatic is the phenomenon of affect regression, which Wurmser (29) observed among addicted patients studied in a psychoanalytic context. He pointed out that when addicts suffer narcissistic injury, they are prone to a precipitous collapse of ego defenses and the consequent experience of intense and unmanageable affective flooding. In the face of such vulnerability, they handle stress poorly and may turn to drugs for relief. This vulnerability can be considered in light of the model of conditioned withdrawal, whereby drug seeking can become an immediate reflexive response to stress, undermining the stability and effectiveness of a patient's coping mechanisms. This can occur quite suddenly in patients who have long associated drug use with their attempts to cope with stress, as illustrated in the following case vignette.

Case 2. In the course of the therapy of an alcoholic lawyer it emerged that his drinking had often been precipitated by situations that threatened his self-esteem. After 6 months of sobriety, he suffered a relapse that was later explained in a session as follows. Immediately before his relapse he had received an erroneous report that his share of the partnership's profits would be cut back, which he took to be evidence of failure. He reported feeling humiliated and then very anxious.

Without weighing the consequences, he went out to purchase a bottle of liquor, returned to his office, and began drinking. He said that he had not thought to control this behavior at the time.

The model of conditioned drug seeking has been applied to development of treatment techniques, to training patients to recognize drug-related cues, and to avert relapse. Annis (17), for example, has used a self-report schedule to assist patients in identifying the cues, situations, and moods that are most likely to lead them to alcohol craving. Marlatt (16) evolved an approach he described as "relapse prevention," whereby patients are taught strategies for avoiding the consequences of the alcohol-related cues they have identified, and a similar conception has been used to extinguish cocaine craving through cue exposure in a clinical laboratory (30).

These approaches can be introduced as part of a single-modality behavioral regimen, but they can also be used in expressive and family-oriented psychotherapy. For example, Ludwig et al. (27) suggested the approach of cognitive labeling, namely, associating drinking cues with readily identified guideposts to aid the patient in consciously averting the consequences of prior conditioning. Similarly, I have described (26) a process of guided recall to explore the sequence of antecedents of given episodes of craving or drinking slips that were not previously clear to the patient. These approaches can be concomitant with an examination of general adaptive problems in an exploratory therapy.

The conditioning approach described here is useful in understanding the relationship between the pharmacology and behaviors associated with drugs of abuse. The test of an explanatory approach, however, is whether it yields options that can be adapted to practice. This means that a stable, ongoing treatment must be secured, one in which cues related to drug seeking can be addressed.

In light of this need for practical application, I will now consider the interpersonal modality necessary to secure an addicted patient's engagement in treatment and compliance. I have called this approach "network therapy" because specific family members and friends are enlisted to provide ongoing support for recovery. This approach will be considered by examining the role of social cohesiveness in securing treatment.

SOCIAL COHESIVENESS AND AMBULATORY CARE

Social cohesiveness is defined as the sum of all forces that act on members of a group to keep them engaged (31). It can be an important factor in binding a patient to the therapy context, even when he or she is inclined to drop out. Dependency on a therapist, affinity for members of a therapy group, or bonds to spouse and children in family therapy are all examples of this phenomenon. In relation to addiction rehabilitation, cohesiveness is particularly important, as it is often the principal vehicle for retaining the addicted patient in

therapy when relapse is threatening. I shall first consider its role in peer-led and established professional programs for addiction. After that I will examine how it can be developed in an office-based modality to secure patients' engagement in addiction rehabilitation.

Peer-Led Programs

In studies of the emergence of cohesiveness in AA and other zealous groups my associates and I have found that when inductees become engaged, they experience an improvement in emotional well-being. This enhanced well-being stabilizes conformity with the group's norms, as compliance is operantly reinforced by a positive affective response to involvement in the group (32, 33). Drug-free therapeutic communities also promote intense relatedness among members as a vehicle for addiction rehabilitation (34), as do the close ties within a given subculture. Alcoholism has also been treated by recourse to group practices in cohesive subcultures. This is seen in peyote rituals in Native American communities in the southwest United States and in *espiritismo* practices among Puerto Rican Americans (35, 36).

AA in particular provides an example of how group cohesiveness can be highly influential in addiction rehabilitation. At AA meetings, reinforcement for involvement is regularly provided as members are given effusive, ritualized approval by the group, both when they speak informally and when they recount their histories at anniversaries of their sobriety. An individual member develops close ties to a member who serves as a sponsor to supervise recovery, and this relationship is a predictor of good outcome (37). On an institutional level as well, the AA approach has been integrated into hospital-based programs, encouraging patients to sustain ties to fellow AA members after discharge (7). In a study of physicians who successfully completed an AA-oriented residential program, my associates and I found that, even 2 years after discharge, they attended more than five AA meetings each week and were in contact with their AA sponsors twice a week (32).

Importantly, AA also illustrates the feasibility of combining strong cohesive ties with cognitive-behavioral techniques. For example, members are inculcated to avoid the "persons, places, and things" that are cues to drinking. They also learn mottos and phrases that serve as cognitive labels (27) for avoidance of problem attitudes and situations. These aspects of the 12-step approach illustrate how the labeling of cues for conditioned withdrawal can be wedded to a social therapy, thereby enhancing the addict's motivation to apply such labeling in avoiding relapse. AA members are reinforced when they discuss the avoidance of cues at AA meetings or with their sponsors in the organization.

Unfortunately, however, many addicted patients reject the option of involvement in AA, and others have been found to drop out after their initial meetings (38). Because of this, there is a strategic advantage in a therapeutic approach that draws on preexisting cohesive ties,

those of family and close friends, as a starting point in treatment. This latter approach can also protect against early dropping out, which might take place in a self-help group setting populated by relative strangers. It can also help the therapist to encourage a reluctant patient to continue attendance at AA meetings.

Professionally Led Treatment

Professionals can also draw on a network of cohesive relationships to enhance the outcome of treatment. For example, an evaluation of the outcome of Speck and Attneave's network therapy for psychotic patients (39) demonstrated that considerable benefit derived from use of existing social ties to family and friends. Enhanced outcome was reported as well when the community reinforcement techniques developed by Hunt and Azrin (40) were augmented by greater social relatedness in a club-like setting (41). Similarly, my colleagues and I (42) effected higher rates of retention and social recovery by integrating a peer-led format into a professionally directed alcohol treatment program.

Not surprisingly, the cohesiveness and support offered by group and family therapy has been found effective in rehabilitating substance-abusing patients. Yalom et al. (43) reported on benefits derived when interactional group therapy was used as an adjunct to recovery techniques. Couples group therapy has also been shown to benefit alcoholics and to diminish the likelihood of treatment dropout (44, 45). Even counseling of spouses of alcoholics in the absence of their alcoholic partners ultimately yielded more effective treatment (46). Observations like these have led experienced clinicians to develop addiction rehabilitation techniques based on expertise in the practice of family therapy and have yielded a number of clinical monographs on the use of established family therapy techniques in addiction (18, 19, 47).

NETWORK THERAPY IN OFFICE PRACTICE

Having examined the need for introducing behavioral techniques and social cohesion into ongoing treatment of the addicted patient, I will now consider the model of network therapy for addiction (20, 48). It offers a pragmatic approach to augmenting conventional individual therapy that draws on these recent advances to enhance the effectiveness of office management.

Couples

A cohabiting couple will provide the first example of how natural affiliative ties can be used to develop a secure basis for rehabilitation. Couples therapy for addiction has been described in both ambulatory and inpatient settings, and good marital adjustment has been found to be associated with a diminished likelihood of dropping out and a positive overall outcome (18, 19, 45, 46, 48–50).

It is recognized, however, that a spouse must be in-

volved in an appropriate way. Constructive engagement should be distinguished from a codependent (51) or overly involved interaction, which is thought to be a problem in recovery. Indeed, couples managed with a behavioral orientation showed greater improvement in alcoholism than those treated with interactional therapy, where attempts were made to engage them in relational change (52). It is therefore important for clinicians to accord each member of the couple an appropriate and differentiated role, so that the spouse is not placed in a position of pressing the patient to comply with treatment. I will therefore consider here a simple, behaviorally oriented device for making use of the marital relationship, namely, working with a couple to enhance the effectiveness of disulfiram therapy.

The use of disulfiram has yielded relatively little benefit overall in controlled trials, when patients are responsible for taking their doses on their own (53). This is largely because this agent is effective only when it is ingested as instructed, typically on a daily basis. Alcoholics who forget to take required doses will likely resume drinking in time. Indeed, such forgetting often reflects the initiation of a sequence of conditioned drug-seeking behaviors.

Although patient characteristics have not been shown to predict compliance with a disulfiram regimen (54), changes in the format of patient management have been found to have a beneficial effect (55). For example, the involvement of a spouse in observing the patient's consumption of disulfiram yields a considerable improvement in outcome (56-58). Patients alerted to taking disulfiram each morning by this external reminder are less likely to experience conditioned drug seeking when exposed to addictive cues and are more likely to comply on subsequent days with the dosing regimen.

The technique (58) also helps in clearly defining the roles in therapy of both the alcoholic and spouse, typically the wife, by avoiding the spouse's need to monitor drinking behaviors she cannot control. The spouse does not actively remind the alcoholic to take each disulfiram dose. She merely notifies the therapist if she does not observe the pill being ingested on a given day. Decisions about managing compliance are then shifted to the therapist, and the couple does not become entangled in a dispute over the patient's attitude and the possibility of secret drinking. By means of this technique, a majority of alcoholics in one clinical trial (59) experienced marked improvement and sustained abstinence over the period of treatment.

A variety of other behavioral devices shown to improve outcome can be incorporated into this couples format. For example, it has been found (60) that scheduling the first appointment for as soon as possible after the initial telephone contact improves outcome by diminishing the possibility of an early loss of motivation. Spouses can also be engaged in history taking at the outset of treatment to minimize the introduction of denial into the patient's representation of the illness (61). The initiation of treatment with such a technique is illustrated in the following case report.

Case 3. A 39-year-old alcoholic man was referred for treatment. Both the patient and his wife were initially engaged by the psychiatrist in a telephone exchange so that all three could plan for the patient to remain abstinent on the day of the first session. They agreed that the wife would meet the patient at his office at the end of the work day on the way to the appointment. This would ensure that cues presented by his friends going out for a drink after work would not lead him to drink. In the session, an initial history was taken from the spouse as well as the patient, allowing her to expand on the negative consequences of the patient's drinking, thereby avoiding his minimizing of the problem. A review of the patient's medical status revealed no evidence of relevant organ damage, and the option of initiating his treatment with disulfiram at that time was discussed. The patient, with the encouragement of his wife, agreed to take his first dose that day, continue under her observation, and then be evaluated by his internist within a few days. Subsequent sessions with the couple were dedicated to dealing with implementation of this plan, and concurrent individual therapy was initiated as well.

Patients who take disulfiram this way have acquired a cognitive label to help them avoid a sudden and unanticipated relapse. The potential efficacy of this approach is illustrated by the reaction of the lawyer described in case 2. He experienced a precipitous collapse of psychological defenses on receiving an incorrect report about his share of the partnership's profits. If he had been taking disulfiram as described here, his knowledge of a potential disulfiram reaction could have alerted him to avoid going out to get a drink. Patients who are maintained with disulfiram as described for an initial year of recovery therefore have the opportunity to deal in therapy with the issues that precipitate craving, without exposing themselves unduly to the threat of relapse. In the lawyer's case, this would have allowed him to address the psychodynamic underpinnings of his job-related anxieties in the therapy, rather than by reflexive drinking.

It is important to clarify certain aspects of engaging a collateral in the treatment, particularly a spouse. Long-standing conflicts between members of an alcoholic couple should not be allowed to interfere with the disulfiram monitoring. For example, the spouse should not be placed in a role in which she must demand compliance. This is why the patient is vested with the responsibility of ingesting the disulfiram so that he is clearly seen by his spouse; her role is only to notify the therapist in a telephone message if she does not see him taking his pill on a given morning. Discussions of compliance per se are therefore initiated by the therapist and not by the spouse. In this way, the role of the spouse as enforcer is eliminated. This is compatible with the approach suggested by Al-Anon, which encourages the spouse to avoid responsibility for managing the other partner's drinking problem.

Larger Networks

In an evaluation of family treatment for alcohol problems reported by the Institute of Medicine, McCrady concluded that "research data support superior out-

comes for family-involved treatment, enough so that the modal approach should involve family members and carefully planned interventions" (1, p. 84). Indeed, the idea of the therapist's intervening with family and friends to start treatment was introduced by Johnson (62) as one of the early ambulatory techniques in the addiction field (14, 15). More broadly, the availability of greater social support to patients has been shown to be an important predictor of positive outcome in addiction (63).

In light of this, it is important to consider what would serve as a useful paradigm for using family and social supports in office treatment. This can be used as well to enhance the stability of the technique for disulfiram observation already described.

The demonstrated utility of directive and behaviorally oriented approaches for preventing relapse might protect against an unstructured exploration of the family as a system. There are, however, two options for stabilizing abstinence: the ecologic and the problem-solving family treatments. The ecologic approach, developed by Minuchin (64) and others, emphasizes the engagement of resources from the patient's family and social environment. It presumes that pathology is embedded in the broader social context and acknowledges that this context must be used to effect recovery. Problem-solving family therapy, developed by Haley (65) and others, relies on an initial assessment of the principal presenting symptom, and subsequent treatment is directed at the problem itself, rather than primarily at restructuring the family relations. By means of these approaches, the therapist can develop an option that parallels the community reinforcement behavioral approach used in multimodality clinics (40).

I reported a positive outcome for this approach with a series of 60 patients treated in network therapy (59). It involved one network session a week for an initial month and subsequent sessions less frequently, typically bimonthly after a year of ambulatory care. Individual therapy was carried out concomitantly once or twice weekly. On average, the networks had 2.3 members, and the most frequent participants were mates, peers, parents, or siblings.

Case 4. Friends of a 46-year-old alcohol-dependent man sought out consultation to secure his abstinence. On the psychiatrist's suggestion, they brought him along with them to a conjoint session, where he avowed that he could stop drinking on his own. An agreement was made among the network members, the patient, and the psychiatrist that they would maintain contact so they could act together in case the patient's suggested approach did not succeed. Two months later, after the patient had required brief hospitalization for detoxification following a relapse into drinking, members of the network prevailed on him to come for treatment. The patient and network members then agreed that he would participate in individual therapy and would meet with the network and psychiatrist at regular intervals. The patient suffered a relapse 6 months later; one of the network members consulted the psychiatrist and then stayed with the patient in his home for a day to ensure that he would not drink. He and the other network members then brought the patient to the psychiatrist's office to reestablish a plan for abstinence.

This case illustrates how members of the network can help to counter the patient's inclination to deny his drinking problem in the initial stages of engagement and during relapse as well. It shows the value of the network in providing the psychiatrist with the means of communicating with a relapsing patient and of assisting in reestablishment of abstinence.

On the other hand, it points to the quandary posed to the clinician who deals with a disorder in which a nonpsychotic patient is subject to uncontrolled, damaging behavior. To what extent should members of the network be encouraged to intervene in the patient's life? Is it proper for the clinician to support their pressing an intoxicated patient to let one of them stay in his house? To exercise proper caution, the therapist must carefully assess the motives and judgment of network members, as well as the patient's capacity to respond positively to their intervention. The therapist must anticipate as much as possible the patient's response to an intervention, both during intoxication and later. Despite a clear need for caution, though, it should be noted that members of AA have for years assumed an active role in helping fellow members terminate relapses into drinking. This aspect of AA underlines the meaningful support that people close to a substance abuser can provide. Incidentally, their support can be instrumental in helping the therapist ensure that the addicted patient is motivated to attend AA meetings and become engaged.

The network is, however, most valuable during conjoint sessions with the patient when it supports the therapist's suggestions for helping the patient avoid relapse. For example, its involvement can be vital in countering the patient's denial of his own vulnerability to relapse. As illustrated in the following case vignette, an effective intervention need involve no more than the network members' providing advice in the therapy session. The weight of the patient's relationship to his own chosen network members and his ability to respond to their efforts to help him are potent tools in securing compliance. In the following case, the network members were instrumental in ensuring that the patient would remove himself from conditioned environmental cues for substance use during the period of early abstinence.

Case 5. A 23-year-old man who had insufflated heroin for a year had recently begun using it intravenously. He abused alcohol and marijuana as well. In a psychiatric consultation that he solicited, he agreed to bring in his uncle, his cousin, and a friend for support and to take naltrexone each day under the observation of the uncle. In the ensuing session with this network, he expressed reluctance to move to his parents' house temporarily to ensure being in a setting that would help him avoid friends who would expose him to regular drinking and marijuana use. After discussing the importance of this added security with his network members and the psychiatrist, he concurred with the consensus that he did need the move temporarily. On the basis of their input, he conceded that it was more important at the moment to avoid the drug cues of his peer group than to insist on independence from his parents.

Sustaining the Network

Yalom (66) has described anxiety-reducing tactics that he used in therapy groups with alcoholics to avert disruptions and promote cohesiveness. These included setting an agenda for the session and using didactic instruction. In the network format, a cognitive framework can be provided for each session by starting out with the patient's recounting events related to cue exposure or substance use since the last meeting. Network members are then expected to comment on this report to ensure that all are engaged in a mutual task with correct, shared information. Their reactions to the patient's report are addressed as well.

Case 6. An alcoholic began one of his early network sessions by reporting a minor lapse in abstinence. This was disrupted by an outburst of anger from his older sister. She said that she had "had it up to here" with his frequent unfulfilled promises of sobriety. The psychiatrist addressed this source of conflict by explaining in a didactic manner how behavioral cues affect vulnerability to relapse. This didactic approach was adopted in order to defuse the assumption that relapse is easily controlled and to relieve consequent resentment. He then led members in planning concretely with the patient how he might avoid further drinking cues in the period preceding their next conjoint session.

This case illustrates the importance of maintaining an appropriate therapeutic milieu in the network sessions. In volunteering to participate, members agree to help the patient but not to subject their own motives to scrutiny. In this, the network format therefore differs materially from the systemic family therapy approach, as it avoids subjecting network members to the demand of addressing their own motives. The didactic or intellectualized approach, as used in case 6, can therefore be helpful in neutralizing excessive anger that may be felt toward the patient without scrutinizing the reasons for a member's anger.

In addition, the patient himself is expected to help maintain amicable relations with network members to protect the supportive milieu. This is made explicit in both network and individual sessions. For example, if a network member is absent for a few sessions, the patient is expected to discuss the matter with that member and resolve any outstanding issues in order to promote the member's return. Any difficulty the patient may experience in carrying out this role is viewed as an issue to be addressed in individual sessions.

The network is therefore conceived of as an active collaboration in which conflicts are minimized to ensure optimal function, as they would be on the work site or in a sports team. When led effectively, members are inclined to be effective team members. They develop a positive transference toward the therapist and are willing to support the therapist's views.

Conditioned Withdrawal and Anxiety

Patients undergoing detoxification from chronic depressant medication often experience considerable anxiety,

even when the dose is reduced gradually (67). The expectation of distress (68), coupled with conditioned withdrawal phenomena, may cause patients to balk at completing a detoxification regimen. In individual therapy alone, the psychiatrist would have little leverage at this point. When augmented with network therapy, however, the added support can be invaluable in securing compliance under these circumstances.

Case 7. A patient elected to undertake detoxification from chronic use of diazepam, approximately 60 mg/day. In network meetings with the patient, her husband, and her friend, the psychiatrist discussed the need for added support toward the end of her detoxification. As her daily dose was brought to 2 mg t.i.d., she became anxious, said that she had never intended to stop completely, and insisted on being maintained permanently with that low dose. Network members supportively but explicitly pointed out that this had not been the plan. She then relented and agreed to the original detoxification agreement, and her dose was reduced to zero over 6 weeks.

The Contingency Contract

Contingency contracting, as used in behavioral treatment (69), stipulates that an unpalatable contingency will be applied if a patient carries out a prohibited symptomatic behavior. Crowley (70) successfully applied this technique to rehabilitating cocaine addicts by preparing a written contract with each patient which stated that a highly aversive consequence would be initiated for any use of the drug. For example, for an addicted physician, a signed letter in which the physician admitted addiction was prepared for mailing to the state licensing board. The approach can be adapted to the network setting as well.

Case 8. A patient regularly attended network and individual therapy sessions and also attended Narcotics Anonymous. Nonetheless, he frequently slipped into cocaine use. In a network session he agreed to random weekly urinalyses and collection of urine samples by his friend, a member of the network. In discussion with network members, he further agreed to prepare a letter to his employer indicating that he was an addict and not suitable to remain on the job. The patient signed an agreement stating that the letter should be mailed by the psychiatrist if any of his weekly urinalyses revealed that he had ingested cocaine. He remained substance free with this regimen over the ensuing year, and his improved status was discussed in network sessions over that time. He continued to be substance free after the contingency was discontinued as well.

Complementing Individual Therapy

Psychotherapeutic approaches have been found to yield improved outcome when combined with certain addiction treatments, such as AA (37), methadone maintenance (71), and cocaine management techniques (72). In the context of network therapy, individual expressive sessions can complement the abstinence orientation of network meetings if the therapist closely attends to conditioned cues for substance use. Once abstinence is stabilized, network sessions can augment

the psychotherapy with support for the patient's general social recovery.

Even after the patient's abstinence is apparently stable, it is important to examine in therapy the patient's thoughts about drinking, dreams related to substance use, and responses to environmental drinking cues. On the one hand, they alert the patient to the need to be aware of the long-term risk of relapse. In addition, they provide revealing clues to ongoing conflicts, which may be apparent only in their expression in the symbolism of addiction.

Although network sessions may be terminated before long-term individual therapy comes to an end, it is essential to make clear that the network members should be available if the patient experiences difficulties in the future, as illustrated in the next case vignette.

Case 9. An alcoholic woman had been seen in network and individual sessions for 16 months and had been abstinent for a year. Because of her stability, a final network session was scheduled with her husband and two friends. Discussion there initially focused on her successful recovery, evidenced by her beginning employment in the previous month. Those present then agreed that any of the network members could contact the therapist if the patient relapsed in the future. The patient herself indicated that she herself would discuss any lapse in abstinence with both the network members and the therapist.

SUMMARY

The model of addictive behavior and office-based treatment presented here deals with the influence of pharmacologically conditioned drinking cues on relapse into substance dependence, and it uses a cognitive-behavioral approach to averting relapse. To engage addicted patients while treatment is applied and to motivate them to overcome the effect of addictive cues, a network of persons close to the patient can be brought into the therapy sessions and augment the individual treatment. Specific network techniques draw on the variety of relationships among the patient, the family, and peers.

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Comparison of Cognitive-Behavioral and Supportive-Expressive Therapy for Bulimia Nervosa

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***Objective:** The authors compared the effectiveness of 4 months (18 sessions) of cognitive-behavioral and supportive-expressive therapy for bulimia. **Method:** Sixty patients obtained from clinical referrals to an eating disorders program who met modified DSM-III-R criteria for bulimia nervosa were randomly assigned to the two conditions. Treatments were delivered in an individual format, on an outpatient basis, by experienced therapists using treatment manuals. The primary outcome measures were self-induced vomiting, binge eating, and attitudes toward body weight and shape, which were assessed by self-report and structured interview. **Results:** Fifty patients completed treatment, 25 in each condition. Both treatments led to significant improvements in specific eating disorder symptoms and in psychosocial disturbances. Supportive-expressive therapy was just as effective as cognitive-behavioral therapy in reducing binge eating. Where treatment differences were found, they favored cognitive-behavioral therapy. Cognitive-behavioral therapy was marginally superior in reducing the frequency of self-induced vomiting; 36% of the patients who received cognitive-behavioral therapy and 12% of those who received supportive-expressive therapy abstained from vomiting in the last month of treatment. Cognitive-behavioral therapy was significantly more effective in ameliorating disturbed attitudes toward eating and weight, depression, poor self-esteem, general psychological distress, and certain personality traits. **Conclusions:** These results moderately favor cognitive-behavioral therapy over supportive-expressive therapy for bulimia nervosa, but follow-up is required to determine the durability of outcome with both modalities. The findings must be interpreted with caution since the selected clinical sample in this study may not represent the bulimia nervosa population.*

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Since the original clinical description of bulimia nervosa by Russell in 1979 (1), there have been numerous reports of psychological and pharmacological treatments for bulimia nervosa. The predominant orientation in psychological treatment trials has been cognitive-behavioral therapy. Despite variation in the actual methods used and in the rigor of study designs, results have indicated that various forms of this intervention can significantly reduce binge eating, vomiting, and associated symptoms and that improvements endure for at least a year after treatment (2, 3).

Only a minority of the treatment studies have used a design aimed specifically at testing the comparative effects of two or more credible treatments, and the results

of these studies have been inconsistent. Three studies (4–6) showed few differences on most dependent measures between cognitive-behavioral therapy and the comparison treatment. The failure of comparative trials to find group differences may relate to the relative importance of nonspecific treatment factors compared to components unique to cognitive-behavioral therapy. Alternatively, it may relate to methodological issues such as inadequate power resulting from small subject groups or failure to adequately distinguish the components of the different treatment conditions. Two more recent studies provide stronger support for the superiority of cognitive-behavioral therapy over other methods. Fairburn et al. (7) compared cognitive-behavioral therapy, a simplified behavioral treatment, and interpersonal psychotherapy. The findings at the end of treatment indicated that, while all three treatments resulted in improvement on measures of psychopathology, cognitive-behavioral therapy was more effective than the other treatments in modifying extreme dieting, self-induced vomiting, and disturbed attitudes toward shape and body weight. In a carefully controlled comparison of imipramine and short-term intensive group treatment that incorporated many components of cognitive-behavioral therapy, Mitchell et al. (8) found that the intensive group treatment was superior to antidepressant treatment alone in modifying eating symptoms; however, both treatments led to significantly greater improvements in eating symptoms and mood disturbance than did placebo treatment. Adding the drug therapy to the group treatment led to additional improvement in measures of anxiety and depression. However, it did not significantly improve outcome related to eating behavior, and it resulted in a higher dropout rate.

To date, several studies have yielded positive findings with psychodynamically oriented treatment (9–13); however, none has involved a comparison with an alternative treatment and all have used such methods as self-monitoring, meal planning, education regarding the untoward effects of dieting, and behavioral contracting, which are generally considered more conceptually aligned with cognitive-behavioral therapy. Thus, it is not clear whether the observed changes in behavior were due to the specific components of the dynamic treatment or to dietary management. Very little improvement in binge eating and vomiting was reported after a 12-week psychodynamically oriented group program that did not include dietary management (14), but the brief group treatment format may not be a sufficiently potent test of the intervention.

The aim of the present study was to compare cognitive-behavioral therapy (4, 7, 15) and a brief psychodynamic therapy (16), both delivered in an individual format, according to specific guidelines, and by experienced therapists. The psychodynamic condition selected for the present study was judged as particularly suitable because it is well conceptualized, manual based, and intended to be brief and it has been described as appropriate for individuals with impulse con-

trol problems. Efforts were made in the current study to use measurement techniques and a time frame for assessments that parallel those in a recent trial by Fairburn et al. (7). A waiting list control group was not used since previous research has indicated that this control condition leads to minimal symptom change. We focused specifically on the magnitude of behavioral and psychological change with these two psychological treatments after 18 individual treatment sessions spaced over a minimum of 4 months.

METHOD

Subjects

The study participants were 60 women who were self-referred or referred by professionals to a hospital eating disorders program and who met the following inclusion/exclusion criteria: 1) the Russell criteria for bulimia nervosa (17) and the DSM-III-R criteria for the disorder with the exception that a minimum average of two binges a week involving *large* amounts of food was not required (criterion A), 2) a minimum of two episodes of vomiting a week for the past month, 3) a minimum duration of illness of 1 year, 4) a present body weight of between 85% and 120% of matched population mean weight (18), 5) age between 18 and 35 years, 6) no concurrent treatment for bulimia nervosa, and 7) written and informed consent to participate. The rationale for the reliance on the rather broad Russell criteria (17) for bulimia nervosa in favor of those specified by DSM-III-R was that there is no consensus on several aspects of the DSM-III-R criteria for bulimia nervosa, such as the definition of a binge and what constitutes a "large amount of food" (19). Several studies have indicated that almost one-half of the episodes labeled by bulimia nervosa patients as "binges" involve fewer than 1,000 calories (20–22). Nevertheless, the amount and type of food consumed during a binge was determined by a standardized interview, the Eating Disorder Examination (23). Four patients (two in each treatment condition) reported no objective episodes of binge eating (consumption of more than 1,000 calories of food not resembling a meal) in the month preceding the assessment interview (they vomited after consuming even small amounts of food), but all reported episodes that met their own subjective definitions of binge eating. The patients reported between 0 and 140 objectively defined binge eating episodes per month (mean=27.5, SD=25.1) and between eight and 154 vomiting episodes a month (mean=42.2, SD=32.6) before the pretreatment assessment.

During the 15-month entry phase of the study, 92 (31.0%) of the 297 patients receiving initial consultations for any form of eating disorder were referred to the present study. The referred patients were assessed by a clinician/research technician who determined whether they met the study criteria; 60 (65.2%) of these patients actually met the inclusion criteria and were entered into the trial.

The patients were stratified according to duration of illness (<3 years and ≥3 years), current weight (86%–110% and >111% of matched population mean weight), and probable history of anorexia nervosa (i.e., adult weight <85% of matched population mean weight), which was derived from information provided on the study intake referral form. They were then randomly assigned by the nonblind study research technician to treatment conditions before the initial assessment interviews. Every attempt was made to adhere to the stratification procedures; however, in a few cases a patient who should have been assigned to one treatment was assigned to the other because therapists in the assigned condition were unavailable to accept a referral at the time. Any patient who dropped out was replaced by the next suitable patient, who was assigned to the same treatment cell, in order to obtain 25 patients who completed each treatment. Previous research indicates that attrition rates of approximately 15% can be expected in treatment studies of bulimia nervosa (2).

Procedure

The nature of the study was explained to prospective participants in an initial meeting, and written informed consent was obtained. Assessments were performed through interviews before treatment (within 3 weeks of the initial assessment), at midtreatment, after treatment, and 3 months, 6 months, and 1 year after the end of treatment by a clinician/research technician not involved in the patients' clinical care. The patients were asked to keep records of the frequency of binge eating, vomiting, laxative abuse, and dieting and to complete the 26-item version of the Eating Attitudes Test (24) throughout the course of treatment as part of the research protocol. The Eating Attitudes Test data were gathered once a week over the first month of treatment and every other week thereafter, and the symptom data were gathered for each week of treatment. The symptom summary forms and Eating Attitudes Test results were placed in a sealed envelope and deposited in a data collection box before treatment sessions. Because the rate of compliance with completing the symptom summary forms decreased markedly after the first 6 weeks of treatment, these data are not presented in the current report. To minimize potential effects of therapist access to study data, the patients were informed that the treatment and research aspects of the study were intended to be kept completely separate and their therapists would not be informed of the results from any of their assessment measures or their progress during treatment. During follow-up the patients were told that their therapists would be given only very general information about their clinical status if it was requested.

At each assessment the patients were weighed, given a structured interview by the study research technician, and then asked to complete a battery of psychometric instruments. They were given instructions regarding the completion of symptom summary forms. The primary outcome measures were frequency of vomiting and

binge eating. Measures of attitudes toward weight and body shape were also considered to be critical in examining the relative effectiveness of the two treatments. Secondary or exploratory measures were aimed at assessing psychological distress, personality features, and social adjustment.

Frequency of vomiting and binge eating were determined before and after treatment with a standardized structured interview, the Eating Disorder Examination (23). Concerns regarding weight, body shape, and eating were also assessed before and after treatment by the drive for thinness, bulimia, and body dissatisfaction subscales of the Eating Disorder Inventory (25), the 26-item version of the Eating Attitudes Test (24), and the Eating Disorder Examination (23).

The psychological measures completed before and after treatment included the remaining subscales of the Eating Disorder Inventory, the SCL-90-R (26), the Borderline Syndrome Index (27), the Rosenberg Self-Esteem Scale (28), the Beck Depression Inventory (29), and the Millon Clinical Multiaxial Inventory (30). The subjects completed the entire Millon inventory, but only the results from the borderline and dysthymia subscales are presented since these subject domains are considered particularly relevant to eating disorders (31).

The Social Adjustment Scale—Self-Report (32) was also administered. Although this scale yields scores for five areas of social functioning, only the aggregate score for all of these areas was used for the data analyses.

A 25-item treatment satisfaction measure, based on the instrument described by Luborsky (16), was developed for the present study. This measure was administered at the posttreatment assessment and evaluated a broad range of factors related to satisfaction with the therapist and with the treatment provided. At the end of treatment, the patients were also asked to evaluate the treatment received in terms of the relative emphasis on 14 general content areas. Items for this measure were generated by the clinicians participating in the study and were based on themes that they judged to have theoretical relevance to one or both treatment conditions.

Treatment Conditions

The subjects were asked to attend 19 individual treatment sessions, each 45–60 minutes in duration, delivered over 18 weeks. The sessions occurred twice a week during the first month, once a week for the next 2 months, and once every other week for the final 6 weeks, in accordance with the model by Fairburn (15). The therapists were 10 experienced clinicians (five with M.D. degrees and five with Ph.D. degrees) who were each recruited for the present study to deliver only the type of treatment that was consistent with his or her usual clinical orientation. This was different from other comparative studies, in which the therapists delivered all available forms of treatment. While this design feature fails to control for potential sources of bias associated with therapist factors, it assures that each therapist is practicing a therapy that is consistent with the thera-

pist's orientation, and it also minimized potential therapist "drift," which may result from delivering divergent forms of treatments. The therapists followed detailed manuals for both treatment conditions and attended weekly supervision meetings to encourage adherence to the treatment protocol when dealing with emergent clinical problems. (As a check on treatment fidelity, a Ph.D. clinician blindly rated 15 randomly selected tapes of therapy sessions from each treatment condition; the clinician was able to correctly classify each tape as representing either cognitive-behavioral therapy or supportive-expressive therapy. Each tape was also rated to determine whether at least three supportive-expressive or cognitive-behavioral therapy components—extracted from the respective manuals—could be identified. Each of the tapes evaluated contained at least three components of the respective treatment, providing further evidence of treatment fidelity.) All therapists were instructed to inform patients that the research component of the study was independent from their treatment and that the therapists would not be informed of the research evaluation at any stage of treatment.

The structure of the protocol for cognitive-behavioral therapy and the overall goals of each stage of treatment generally followed the manual described by Fairburn (15), supplemented by our own adaptation of cognitive-behavioral therapy principles for eating disorders (33–35). These principles were originally derived from techniques developed by Beck (36) and colleagues for the treatment of depressive and anxiety disorders. The patients were supplied with self-monitoring forms and asked to keep records of all food and liquid ingested; episodes of binge eating, vomiting, laxative abuse, and other weight-losing behaviors; and feelings and thoughts concerning eating.

The supportive-expressive therapy used the treatment manual by Luborsky (16), supplemented by psychodynamic writings on eating disorders (37–39). The style of the supportive-expressive therapy was nondirective, and the emphasis was on listening to the patient and helping identify problems and their solutions. No specific advice was to be given at any stage. The therapist was instructed to assume a facilitative role and allow the patient to retain responsibility for change. Adapted to eating disorders, the supportive-expressive therapy approach was based on the assumption that the eating symptoms serve a functional role by disguising underlying interpersonal problems.

Although information was gathered on present eating symptoms and each patient was given an information booklet outlining the dangers of binge eating, self-induced vomiting, and purgative abuse, the therapists were instructed to "be careful to avoid giving patients specific advice" and were told that if questions were asked by the patient, the "question should be reflected back."

Data Analysis

The overall effects of the two treatment conditions were evaluated for each outcome variable by using a two-way

repeated measures analysis of variance (ANOVA) with treatment group and time as the factors. The differences between the two treatment effects were determined by an analysis of covariance (ANCOVA) for each outcome variable with the pretreatment patient scores on the respective measures as the covariates.

Frequency of vomiting and binge eating as assessed by self-report and interview were considered the principal bases for interpreting the comparative efficacy of the two treatments. Nevertheless, additional data are presented for measures of attitudes toward weight and shape and for secondary measures tapping a broad range of psychosocial areas of functioning. Exploratory analyses of a relatively large number of outcome measures were considered to be an important adjunct to formal hypothesis testing in the current study because of their potential for advancing the understanding of meaningful treatment variables (40). It was decided not to reduce the number of outcome measures by using data reduction procedures, such as principal component analysis, in spite of the problems imposed by the unfavorable subject-to-variable ratio, since this would result in difficulty in interpreting factors in relation to normative data and change scores for instruments widely used in previous research. Changes in scores on these instruments are of substantial interest to both clinicians and researchers despite the probable redundancy of some measures and the potential type I and type II errors due to multiple comparisons. While nonadjusted probabilities are presented in the tables to facilitate the examination of the pattern of group differences across all measures, probabilities adjusted to control for the family-wise error rate were calculated and are also included in the tables.

RESULTS

Dropouts and Completers

Ten patients (five patients from each treatment condition) withdrew before the end of treatment and were not followed. The proportional distribution of dropouts across the two treatment conditions reduces, but does not eliminate, the possibility that the posttreatment comparisons were biased in favor of either of the treatment conditions.

The representativeness of the patients who completed treatment was evaluated by collapsing the completers and dropouts across both treatment conditions and comparing these two groups on pretreatment age, weight and weight history variables, duration of illness, target eating symptoms, and scores on Eating Disorder Inventory subscales, Eating Attitudes Test subscales, SCL-90-R subscales, Borderline Syndrome Index, Beck Depression Inventory, Social Adjustment Scale total, and Rosenberg scale. The dropouts did not differ significantly from the completers on any of these variables except that their maximum adult weight, expressed as a percentage of matched population mean weight, was

higher than that of the completers ($t=3.27$, $df=56$, $p<0.002$), and they showed more disturbance than the completers on the Beck Depression Inventory ($t=2.34$, $df=58$, $p<0.03$), Eating Attitudes Test oral control subscale ($t=2.79$, $df=56$, $p<0.007$), and SCL-90-R somatization subscale ($t=2.58$, $df=58$, $p<0.02$). These findings, along with the sizable proportion of referred subjects who were not actually entered into the trial, suggest that the findings from the present study may not be representative of all bulimia nervosa patients requesting treatment for the disorder.

The remaining findings reported in the present study relate to the 50 patients who completed treatment and include those who, by mutual consent, terminated treatment early because they had met the treatment objectives (this includes five patients from the cognitive-behavioral therapy condition who terminated after sessions 9, 10, 12, 13, and 18, respectively, and five patients receiving supportive-expressive therapy who terminated treatment at session 18 rather than 19). Demographic data and clinical features for these 50 patients are summarized in table 1. Before treatment there were no significant differences between the cognitive-behavioral and supportive-expressive treatment groups in age, height, actual weight, weight expressed as percentage of matched population mean weight, duration of illness (almost 6 years), previous treatment for an eating disorder (45.5% versus 52.2%), or history of an adult weight below 80% of matched population mean weight (28.0% in each group). The only psychometric instrument on which there was a significant pretreatment group difference was the maturity fears subscale of the Eating Disorder Inventory, on which the subjects who received supportive-expressive therapy had a higher mean initial score ($t=2.32$, $df=47$, $p<0.03$). The supportive-expressive therapy group also had a somewhat higher mean pretreatment score on the Borderline Syndrome Index ($F=1.80$, $df=47$, $p<0.08$).

Content Areas Discussed During Treatment

At the posttreatment assessment, the patients were asked to rate on a 14-item, 6-point ("never" to "always") Likert scale "how often you and your therapist discussed" specific content areas during treatment. The differences between the treatment groups were consistent with the theoretical expectations for the respective treatments and provided some reassurance that the treatment received was consistent with the intended model.

There were six significant differences and two nearly significant differences ($p<0.09$) between the ratings of 14 content areas by the subjects in the two treatment conditions, and all of these were in the expected direction. The subjects who received cognitive-behavioral therapy gave higher ratings than did the supportive-expressive therapy subjects for discussions of "preoccupation with weight and shape" ($t=9.68$, $df=40$, $p<0.0001$), "bingeing" ($t=5.01$, $df=40$, $p<0.0001$), "preoccupation with food" ($t=4.53$, $df=40$, $p<0.0001$),

TABLE 1. Baseline Features of Bulimic Patients Receiving Cognitive-Behavioral Therapy (N=25) or Supportive-Expressive Therapy (N=25)

Characteristic	Cognitive-Behavioral Therapy		Supportive-Expressive Therapy	
	Mean	SD	Mean	SD
Age (years)	23.7	4.4	24.6	4.0
Height (in)	65.6	3.0	66.1	2.5
Actual weight (lb)	126.4	16.4	126.6	13.1
Weight as percentage of matched population mean weight				
Current	95.3	9.8	94.9	7.9
Maximum	108.6	9.9	111.8	12.7
Minimum	84.3	10.0	82.9	8.7
Duration of illness (months)	71.8	47.6	71.2	40.2

"vomiting" ($t=3.60$, $df=40$, $p<0.001$), and "specific strategies for overcoming your eating problems" ($t=6.90$, $df=40$, $p<0.0001$), and there was a nearly significant difference for "the causes of your eating problems" ($t=1.86$, $df=40$, $p<0.07$). The ratings of the subjects who received supportive-expressive therapy were significantly higher for "your relationship with your parents when you were a child" ($t=3.76$, $df=40$, $p<0.01$), and the difference was nearly significant for "your relationship with your therapist" ($t=1.89$, $df=40$, $p<0.07$). There were no significant differences between treatments on "your relationship with your parents," "your relationship with men," "your beliefs and attitudes in general," "your feelings and emotions in general," "your self-esteem," and "your mood."

Symptom Measures

The two-way repeated measures ANOVAs indicated that there was a main effect of time for all outcome variables, indicating that the treatments had a significant effect on the symptom areas measured.

Table 2 presents the mean frequencies of vomiting and objective binge eating, as determined by interview, for both treatment conditions. The two treatments were equally effective in their impact on binge frequency, but the group differences in vomiting frequency, in favor of the cognitive-behavioral therapy group, approached significance. There was an 81.9% reduction in vomiting frequency from pre- to posttreatment in the cognitive-behavioral therapy group, compared to a 62.1% reduction in the supportive-expressive therapy group. At the end of treatment, most patients had improvements in vomiting frequency of at least 50% (92.0% of the patients receiving cognitive-behavioral therapy and 68.0% of the patients receiving supportive-expressive therapy); however, only nine (36.0%) of the cognitive-behavioral therapy patients and three (12.0%) of the patients receiving supportive-expressive therapy had been abstinent from vomiting for the 28 days preceding the interview evaluation. Table 2 also presents the results of the ANCOVAs for the Eating Attitudes Test; cognitive-behavioral therapy led to significantly greater

TABLE 2. Eating Symptoms and Scores on the Eating Attitudes Test and Eating Disorder Examination for Bulimic Patients Before and After Cognitive-Behavioral or Supportive-Expressive Therapy

Measure	Cognitive-Behavioral Therapy			Supportive-Expressive Therapy			ANCOVA ^a		
	N	Mean	SD	N	Mean	SD	F	df	p
Vomiting episodes in last 28 days	25			25					
Pretreatment		41.4	38.7		44.1	30.5			
Posttreatment		7.5	13.5		16.7	18.5	3.72	1, 49	0.06
Binge eating episodes in last 28 days	23			23					
Pretreatment		26.3	30.2		31.1	20.3			
Posttreatment		7.1	14.1		9.6	11.0	0.08	—	n.s.
Eating Attitudes Test (26-item)	25			23					
Dieting									
Pretreatment		20.6	8.6		19.7	7.7			
Posttreatment		6.8	5.9		12.5	9.5	7.76	1, 48	0.008
Bulimia and food preoccupation									
Pretreatment		11.2	4.3		10.9	4.0			
Posttreatment		2.0	3.7		4.9	4.5	6.60	1, 48	0.01
Oral control									
Pretreatment		2.9	2.9		2.8	3.6			
Posttreatment		1.6	1.4		1.3	1.9	0.31	—	n.s.
Total									
Pretreatment		34.7	12.7		33.2	11.6			
Posttreatment		10.4	9.1		18.7	14.1	7.25	1, 48	0.01
Eating Disorder Examination	25			25					
Dietary restraint									
Pretreatment		3.7	1.3		3.2	1.5			
Posttreatment		1.5	1.7		2.5	1.6	7.37	1, 47	0.009
Attitudes toward shape									
Pretreatment		3.3	1.4		3.6	1.0			
Posttreatment		2.0	1.3		2.9	1.1	5.77	1, 47	0.02
Attitudes toward weight									
Pretreatment		2.4	1.4		2.9	1.1			
Posttreatment		1.6	1.2		2.4	1.1	3.59	1, 47	0.07

^aThe family-wise error rate for 12 comparisons (all variables in table 2 and the first three subscales of the Eating Disorder Inventory in table 3) is 0.6 for $p=0.05$, 0.12 for $p=0.01$, and 0.012 for $p=0.001$.

improvements in the scores on two Eating Attitudes Test subscales and on the total score.

Table 2 also contains findings for the same Eating Disorder Examination subscales reported by Fairburn et al. (7). In the current study, cognitive-behavioral therapy was significantly superior to supportive-expressive therapy in effects on dietary restraint and shape concerns, and there was a nearly significant difference in the score on the weight concerns subscale. Not reported in the table is the failure to achieve group differences on the Eating Disorder Examination bulimia subscale and the significant group difference, favoring cognitive-behavioral therapy, on the eating concerns subscale ($F=5.97$, $df=1, 47$, $p<0.02$).

Presented in table 3 are the results from the ANCOVAs for the Eating Disorder Inventory subscales. There was a significant group difference on the bulimia subscale and a nearly significant difference on the drive for thinness subscale; both indicated superiority of cognitive-behavioral therapy. There was a significant group effect for the maturity fears subscale of the Eating Disorder Inventory; however, as indicated earlier, the group that received supportive-expressive therapy had higher pretreatment and posttreatment scores on this subscale than the cognitive-behavioral therapy group.

A repeated measures Group by Time ANOVA indicated that the patients experienced a significant weight increase during treatment ($F=23.60$, $df=1, 47$, $p<0.0001$), and the Group by Time interaction was nearly significant ($F=3.37$, $df=1, 47$, $p<0.08$), reflecting the fact that the cognitive-behavioral therapy group gained more weight during treatment than the supportive-expressive therapy group (mean weight gain for the cognitive-behavioral therapy group was 6.6 lb, or 3.0 kg, to 100.4% of matched population mean weight; mean weight gain for the supportive-expressive therapy group was 3.0 lb, or 1.4 kg, to 97.6% of matched population mean weight).

Table 4 summarizes data for both treatment groups on the Beck Depression Inventory, the Borderline Syndrome Index, the borderline and dysthymia scales of the Millon Clinical Multiaxial Inventory, the global SCL-90-R, the Rosenberg Self-Esteem Scale, and the total Social Adjustment Scale. The number of subjects varies among the different measures owing to the facts that some patients did not complete certain psychometric instruments and the decision to administer the Millon Clinical Multiaxial Inventory was made after the study had begun. The ANCOVAs showed better results for the cognitive-behavioral therapy condition, including

significant posttreatment differences on the Beck Depression Inventory, the borderline and dysthymia scales of the Millon Clinical Multiaxial Inventory, the global SCL-90-R, and the Rosenberg scale. Although not presented in table 4, there were also significant group differences in favor of cognitive-behavioral therapy on the schizotypal ($F=5.79$, $df=1, 35$, $p<0.02$), anxiety ($F=7.55$, $df=1, 35$, $p<0.01$), and somatoform ($F=12.28$, $df=1, 35$, $p<0.001$) subscales of the Millon Clinical Multiaxial Inventory.

Treatment Satisfaction

Satisfaction with treatment was rated by the patients at the posttreatment assessment with a 25-item scale adapted from Luborsky (16). This scale tapped a number of elements of treatment and had a reliability coefficient (α) of 0.95 for the patients completing treatment. On the basis of monthly frequency of vomiting at the end of treatment, the patients were classified as having either good/moderate outcome (≤ 4 episodes per month) or poor outcome (>4 episodes per month). A Treatment by Outcome Group ANOVA for treatment satisfaction revealed a significant interaction ($F=5.72$, $df=1, 46$, $p=0.02$). Cognitive-behavioral therapy patients with good outcomes were significantly ($F=9.55$, $df=3, 46$, $p<0.0001$) more satisfied with treatment than were cognitive-behavioral therapy patients with poor outcomes or supportive-expressive therapy patients with either good or poor outcomes.

DISCUSSION

The major aim of the current study was to compare cognitive-behavioral therapy to a credible alternative matched on nonspecific therapeutic factors but not using methods considered integral to cognitive-behavioral therapy for bulimia nervosa. Both cognitive-behavioral therapy and supportive-expressive therapy led to significant improvements in specific eating disorder symptoms and in a broad range of measures of psychosocial functioning. Although previous comparative research has shown that treatments other than cognitive or behavioral therapies can lead to improvements in eating symptoms (4, 6), these results have been difficult to interpret because of a blurring between treatment conditions. This makes the supportive-expressive therapy findings in the current study, along with the findings of another recent study (7), of particular interest, since they suggest that treatments which do not specifically focus on eating behavior or concerns about eating or weight (unlike cognitive-behavioral therapy) are able to lead to significant improvements in these areas. In the current study, supportive-expressive therapy was just as effective as cognitive-behavioral therapy in reducing episodes of binge eating. This is consistent with the findings of the Fairburn et al. (7) study, in which interpersonal therapy compared favorably to cognitive-behavioral therapy in reducing binge eating episodes.

TABLE 3. Scores on Subscales of the Eating Disorder Inventory for Bulimic Patients Before and After Cognitive-Behavioral (N=25) or Supportive-Expressive (N=24) Therapy

Subscale of Eating Disorder Inventory	Score				ANCOVA (df=1, 48) ^a	
	Cognitive-Behavioral Therapy		Supportive-Expressive Therapy			
	Mean	SD	Mean	SD	F	p
Drive for thinness						
Pretreatment	14.3	4.4	14.1	5.2	3.63	0.06
Posttreatment	5.9	6.3	9.4	6.8		
Bulimia					10.33	0.002
Pretreatment	11.6	4.9	10.2	6.2		
Posttreatment	2.2	3.9	4.8	4.5		
Body dissatisfaction					0.48	n.s.
Pretreatment	15.5	8.4	16.7	8.0		
Posttreatment	11.7	9.0	13.7	7.5		
Ineffectiveness					1.60	n.s.
Pretreatment	8.6	6.3	10.0	6.9		
Posttreatment	4.9	7.0	7.7	6.2		
Perfectionism					1.56	n.s.
Pretreatment	6.8	4.5	8.0	3.5		
Posttreatment	4.4	3.7	6.3	4.2		
Interpersonal distrust					0.22	n.s.
Pretreatment	5.0	4.1	5.0	4.0		
Posttreatment	3.0	3.1	3.3	3.1		
Interoceptive awareness					1.50	n.s.
Pretreatment	8.7	6.1	9.9	4.6		
Posttreatment	2.9	4.7	4.8	4.2		
Maturity fears					0.60	n.s.
Pretreatment	2.6	2.5	5.0	4.6		
Posttreatment	1.3	1.5	3.2	4.2		

^aThe family-wise error rate for eight comparisons is 0.4 for $p=0.05$, 0.08 for $p=0.01$, and 0.008 for $p=0.001$.

A key finding in this comparative trial was that, where treatment differences did exist, they favored the cognitive-behavioral therapy intervention. Cognitive-behavioral therapy was marginally superior ($p<0.06$) to supportive-expressive therapy in reducing vomiting frequency and also led to greater improvements in most measures of concern about eating and weight, symptom areas that are considered specific to bulimia nervosa. While the findings favoring cognitive-behavioral therapy in modifying vomiting frequency are similar to those of an earlier comparison of cognitive-behavioral therapy and interpersonal therapy (7), the overall percentage reduction in this symptom following cognitive-behavioral therapy in the current study (81.9%) and the proportion of patients who were abstinent from vomiting following cognitive-behavioral therapy (36.0%) are somewhat less impressive than the findings by Fairburn et al. (95% and 47%, respectively). These differences may be due to a number of factors, such as use of different selection criteria in the two studies (i.e., the presence of vomiting was required in the current study but was present in only 72% of the patients in the Fairburn et al. study), differences in the referral base for the two studies (the patients in the current study were selected

TABLE 4. Psychological Functioning of Bulimic Patients Before and After Cognitive-Behavioral or Supportive-Expressive Therapy

Measure	N	Cognitive-Behavioral Therapy		N	Supportive-Expressive Therapy		ANCOVA ^a		
		Mean	SD		Mean	SD	F	df	p
Beck Depression Inventory	25			24					
Pretreatment		16.8	9.9		18.7	9.4			
Posttreatment		7.5	10.6		13.4	9.5	4.05	1, 48	0.05
Borderline Syndrome Index	22			22					
Pretreatment		16.2	11.9		22.9	11.2			
Posttreatment		11.5	11.9		16.4	10.1	0.04	—	n.s.
Rosenberg Self-Esteem Scale	24			23					
Pretreatment		25.0	5.7		23.7	5.3			
Posttreatment		29.4	6.2		25.6	5.2	4.76	1, 46	0.03
SCL-90-R	25			23					
Pretreatment		1.1	0.7		1.3	0.6			
Posttreatment		0.6	0.7		1.0	0.6	5.10	1, 47	0.03
Social Adjustment Scale—Self-Report	20			21					
Pretreatment		2.2	0.5		2.2	0.5			
Posttreatment		1.9	0.5		2.1	0.5	1.98	—	n.s.
Millon Clinical Multiaxial Inventory	16			20					
Borderline subscale									
Pretreatment		73.4	17.9		75.0	13.3			
Posttreatment		56.8	17.4		73.7	20.6	9.06	1, 35	0.005
Dysthymia subscale									
Pretreatment		85.1	17.4		89.2	15.4			
Posttreatment		65.6	18.3		88.1	16.8	15.44	1, 35	0.0001

^aThe family-wise error rate for seven comparisons is 0.35 for $p=0.05$, 0.07 for $p=0.01$, and 0.007 for $p=0.001$.

from patients referred to a specialized tertiary eating disorder center, whereas there were no competing treatment centers in the Fairburn et al. study), or the relative ineffectiveness of the cognitive-behavioral therapy offered in the current study. The relatively low rates of abstinence in the cognitive-behavioral therapy condition demonstrate that this treatment, delivered over the short term, is not curative for most bulimic patients seeking treatment at a tertiary care center specializing in eating disorders. Nevertheless, the percentage reduction in vomiting and the abstinence rates achieved in the current study are consistent with the median rates of improvement seen in other studies of the efficacy of cognitive-behavioral therapy (2).

The results for vomiting were paralleled by changes in measures of concern regarding weight, shape, and eating. Both treatment groups showed significant improvement, and at the end of treatment the group differences favored cognitive-behavioral therapy. The results on the Eating Attitudes Test are consistent with those of earlier comparative studies (4, 7), indicating that cognitive-behavioral therapy is superior to alternative forms of treatment in ameliorating attitudes and behaviors characteristic of eating disorders. The improvements in scores on the Eating Disorder Inventory in the current study are comparable to those reported in the study of drug treatment and group therapy by Mitchell et al. (8). The changes on the Eating Disorder Examination and the Eating Attitudes Test were similar to those reported by Fairburn et al. (7). This is reassuring to the degree that it confirms the clinical sensitivity of these instruments in detecting change.

Perhaps of greater importance than the change in eating symptoms in the current study is the superiority of cognitive-behavioral therapy in promoting changes on measures of depression, self-esteem, personality features, and general psychopathology, since it could be argued that these areas, rather than eating symptoms, represent the targets of change for supportive-expressive therapy. These improvements may be attributed to direct therapeutic effects of cognitive-behavioral therapy on these symptom areas, or they may be simply a reflection of the inverse association between chronic dietary chaos and psychological disturbance observed in bulimia nervosa patients (41, 42). Nevertheless, the relative superiority of cognitive-behavioral therapy in promoting improvement across a number of psychological dimensions contradicts the notion that changes in eating symptoms are unrelated to more fundamental areas of psychological distress (43).

The findings from the current study are limited by several factors. First, the cognitive-behavioral therapy and supportive-expressive therapy were delivered by different therapists, in contrast to earlier studies, in which the compared treatments were delivered by the same therapists. Cognitive-behavioral therapy received significantly higher patient satisfaction ratings than supportive-expressive therapy but only when there was a positive treatment outcome. Thus, while the therapists providing cognitive-behavioral therapy were not uniformly evaluated in a positive manner, the observed differences in treatment outcome in the current study could be due to other systematic sources of bias related to general therapist variables. Second, treatment fidelity

and quality were not assessed for all sessions, leaving open to question whether the specified treatments were competently executed. Although by no means conclusive, some reassurance on these points comes from the following: 1) tape ratings for a subset of sessions indicated that the treatment conditions could be distinguished, 2) patient ratings of treatment content were consistent with theoretical predictions, 3) the treatments were manual based, 4) they were delivered by experienced therapists practicing in their preferred orientations, and 5) the therapists were supervised by senior clinicians. Finally, the conclusions regarding the efficacy of the treatments evaluated in this trial are limited by the absence of follow-up data. While most previous research on psychological treatment of bulimia nervosa has indicated that improvements at the end of treatment are maintained or augmented during the first year of follow-up (2), there is no assurance that this pattern would extend to the groups evaluated in the current study. We are currently gathering follow-up data on these treatments.

At this time little is known about the active ingredients of treatment and the predictors of response to treatment. Moreover, the impact of nonspecific treatment factors on outcome in bulimia nervosa has remained largely unexplored. Several studies (44–47) have evaluated specific components of cognitive-behavioral therapy by using dismantling designs and have begun to clarify the picture regarding the active ingredients of treatment; however, the ideal elements of treatment are not yet evident. Indeed, the “optimal treatment” is probably not generalizable to all patients with bulimia nervosa. Future refinements may point to sequencing or stepwise approaches to treatment tailored to meet the individual needs of members of the heterogeneous patient population (48). This would involve initially providing patients with an educationally oriented treatment (35) that may benefit those who have a more benign variant of bulimia nervosa, followed by more intensive interventions for those who are resistant to change. Of interest in this regard is a comparison of the cognitive-behavioral therapy results in this study with our brief educational treatment (49), which indicated that on several important outcome indexes the two treatments were equally effective with the least symptomatic 25%–45% of patients. Decisions regarding the probable efficacy of the various treatment options are clearly influenced by an understanding of predictors of outcome; however, few studies have carefully evaluated the range of psychosocial and biological factors that may be associated with outcome. This remains a potentially fruitful area for future research.

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HIV Seroprevalence Among Homeless Patients Admitted to a Psychiatric Inpatient Unit

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Objective: This study was conducted to determine the seroprevalence of HIV-1 antibodies among hospitalized homeless mentally ill patients. **Method:** From December 1989 through May 1991 the authors collected discard blood samples from patients consecutively admitted to a psychiatric unit designated for the care of severely mentally ill persons removed from the streets of New York City. The blood samples were tested for HIV-1 antibodies, and the results were analyzed for associations with age, gender, ethnicity, male homosexual activity, and use of injected drugs. **Results:** The HIV seroprevalence was 6.4% (13 of 203 samples). Patients between ages 18 and 39 accounted for 51.2% of the admissions and 84.6% of the 13 positive results, a seroprevalence of 10.6% for this subsample. Patients under age 40 were more than six times as likely to test positive for HIV antibodies as those 40 or over. Ethnicity did not predict seropositivity. Women were as likely as men to be infected. Although clinicians had noted high-risk behavior on the charts for only three (23.1%) of the 13 positive cases, a recorded history of use of injected drugs was associated with a 6.5-fold greater risk of HIV seropositivity. **Conclusions:** One in every 16 patients admitted to the special unit was HIV positive. Age under 40 and use of injected drugs were strongly associated with seropositivity. Because information on high-risk behavior was infrequent, the reasons for younger patients' greater risk are unclear. The homeless mentally ill require outreach efforts to reduce the risk of acquiring or transmitting HIV.

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The effects of the HIV epidemic on persons who are homeless and mentally ill have not been adequately investigated. Few reports on the prevalence of HIV infection among the homeless have been published, and there have been none on the prevalence among the homeless mentally ill.

Estimating the prevalence of HIV among persons who are both mentally ill and homeless is difficult, as they constitute a "hard to reach" population (1). The homeless mentally ill are likely to be chronically disaf-

filiated from social services (2), may be too disorganized to gain access to the mental health system (3), and appear to have little contact with outpatient medical settings (4). Moreover, the homeless mentally ill may not go to public shelters, which often provide on-site medical and psychiatric evaluation. A recent study found evidence of underutilization of shelters by the mentally ill living in public spaces. Of 177 persons removed from New York City streets and admitted to Bellevue Hospital for acute psychiatric care, 42% had not used public shelters during the year before admission (E. Susser, personal communication, Oct. 16, 1991).

The prevalence of HIV infection among the severely mentally ill in New York City is distressingly high. We previously reported a 5.5% seroprevalence of HIV among patients admitted to two public psychiatric hospitals in New York City (5). At a private, voluntary hospital providing acute care in New York City, 7.1% of psychiatric inpatients were seropositive (6). Severe mental illness may increase the risk of HIV infection by contributing to indiscriminate sexual activity or drug use at times of decompensation (7-10). Prevalent substance abuse and exchanging sex for drugs, money, or

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food put persons with severe mental illness at increased risk (8, 11). Substance abuse may be even more prevalent among the homeless than among the domiciled mentally ill (12).

Most evidence about HIV infection among the homeless is indirect, being derived from studies which examine medical conditions that may be associated with HIV rather than the presence of HIV antibodies (13–15). Public interest in the few studies of the prevalence of HIV among the homeless has been intense. A reported HIV prevalence of 66% among 107 shelter users attending an on-site medical clinic was widely disseminated by the media and caused international alarm (R.A. Torres et al., V International Conference on AIDS, 1989). By contrast, a survey of 4,383 runaway adolescents in four states showed that 4.1% were HIV positive (R.L. Stricof et al., VI International Conference on AIDS, 1990). The striking disparity between these prevalence estimates may reflect methodologic differences as much as differences in HIV prevalence between subgroups of the homeless.

In October 1987 New York City established a link between the Homeless Emergency Liaison Project and a continuum of specialized psychiatric services, including an acute care hospital unit and an extended care state hospital ward designated for care of the homeless. The Homeless Emergency Liaison Project is a mobile outreach team that evaluates homeless men and women living on the streets who exhibit psychiatric symptoms. Designated psychiatrists are authorized to order involuntary transport to a psychiatric emergency room of persons who meet commitment criteria broadly interpreted to include grave disability as a form of dangerousness to self (16). Patients appropriate for admission are initially treated at Bellevue Hospital (17). Patients who cannot be discharged from Bellevue Hospital within approximately 28 days are transferred to a unit at Creedmoor Psychiatric Center created for extended care of the homeless mentally ill (17, 18). During the first year of operation of this project for the homeless, 67% of the patients admitted to Bellevue Hospital were transferred to the Creedmoor unit (19). This article reports the results of a seroprevalence study conducted among severely ill psychiatric patients admitted for extended care to the Creedmoor unit for the homeless.

METHOD

We tracked every patient between the ages of 18 and 59 years consecutively admitted to the unit for the homeless during the 18-month period from December 1989 through May 1991. Following current federal regulations for anonymous sampling (20), we obtained discard blood samples from blood drawn for routine purposes. Since samples could not be obtained for every patient at the time of admission because of refusal to permit routine blood workups, discharge of the patient before routine blood drawing, or an insufficient quantity of blood, we used any discard sample of blood

drawn during the study period. We kept a roster of every patient admitted so as to obtain only a single sample of blood for each eligible patient, even if that patient had more than one admission or blood drawing during the study. This roster was not linked to HIV test results and was destroyed when all samples had been obtained.

Patients' chart number, date of birth, admission diagnosis, and number of previous psychiatric hospitalizations were obtained from their charts and recorded on the top half of a two-part form in order to obtain profiles of the eligible patients that were unlinked to their HIV status. Information on age range, gender, ethnicity, use of injected drugs, and homosexual activity among the men was obtained from the charts and recorded on the lower half of the form. Because of the small number of Asian patients admitted, Asians were grouped with Caucasians to avoid the possibility of identifying any HIV-positive Asian patient. After demographic and risk data had been obtained, the patient-identification (upper) section of the form was removed, and a six-digit bar code number was affixed to the lower section to link the blood sample with the demographic and risk data.

Patients whose blood was sampled and patients whose blood was not obtained were not significantly different in diagnosis ($\chi^2=1.71$, $df=3$, $p=0.63$) or number of previous hospitalizations ($t=0.56$, $df=17.01$, $p=0.59$). As a group, the 209 patients whose blood was sampled had the following clinical diagnoses: schizophrenia ($N=103$, 49.3%), schizoaffective disorder ($N=90$, 43.1%), affective disorders ($N=7$, 3.3%), and other psychotic disorders ($N=9$, 4.3%). No patient was given a diagnosis of AIDS dementia. The number of known previous psychiatric hospitalizations was as follows: no hospitalizations, 25 patients (12.0%); one hospitalization, 19 (9.1%); two to five hospitalizations, 122 (58.4%); more than five hospitalizations, 43 (20.6%).

There were no significant differences in age or gender between the patients whose blood was obtained and those whose blood was not sampled. However, black and Hispanic patients were represented somewhat less than Caucasian and Asian patients among those whose blood was collected compared to those whose blood was not collected. Blood was not obtained for 2.1% of the Caucasian/Asian group, 8.7% of the black patients, and 15.4% of the Hispanic group ($\chi^2=7.03$, $df=2$, $p=0.03$).

Blood samples were stored, frozen, and sent in batches of at least 25 to the New York Blood Center for HIV-1 antibody testing. Specimens that were reactive on enzyme immunoassay were tested by the Western Blot assay and then classified according to current recommendations of the Centers for Disease Control (21). Specimens were considered positive when antibodies to two of the following were detected: p24, gp41, and gp120/160. The presence of any single band on the Western Blot constituted an indeterminate result. The anonymous sampling did not permit obtaining and testing a second specimen from patients with indeterminate results on the first test to clarify their HIV status.

TABLE 1. Association of HIV Infection With Demographic Characteristics, Homosexual Activity Among Men, and Use of Injected Drugs^a

Variable	Patients Tested	HIV-Positive Patients		Unadjusted Odds Ratio		Adjusted Odds Ratio ^b		p
		N	%	Odds Ratio	95% Confidence Interval	Odds Ratio	95% Confidence Interval	
Age (years)				5.74	1.24–26.58	6.31	1.28–31.07	0.02
18–39	104	11	10.6					
40–59	99	2	2.0					
Ethnicity ^c				1.86	0.55–6.25	—	—	—
Black	93	6	6.5					
Hispanic	20	3	15.0					
Caucasian and Asian	90	4	4.4					
Sex				1.32	0.35–5.00	—	—	—
Male	146	10	6.8					
Female	57	3	5.3					
Use of injected drugs				4.88	1.17–20.33	6.67	1.42–31.28	0.02
Identified	14	3	21.4					
Not identified	189	10	5.3					
Homosexual activity among men ^d				1.57	0.18–13.78	—	—	—
Identified	10	1	10.0					
Not identified	136	9	6.6					
Any risk factor				2.70	0.68–10.67	—	—	—
Identified	22	3	13.6					
Not identified	181	10	5.5					
Total	203	13	6.4					

^aOf the 209 individuals whose blood was tested for HIV infection, the results for six were indeterminate; therefore, data on 203 individuals were used in this analysis.

^bThe final logistic regression model included age, ethnicity, and use of injected drugs.

^cComparison groups for calculating the odds ratios for ethnicity were black and Hispanic versus Caucasian and Asian.

^dN=146, as this question pertains to male patients only.

The clinical characteristics of the patients, including admission diagnosis and number of previous psychiatric hospitalizations, constituted the group profile of the patients whose blood was sampled, unlinked to the blood specimens. Subsample seroprevalence was derived from blood test results linked with patient demographic characteristics and two HIV risk behaviors. All data were measured at either a nominal or an ordinal level; therefore, contingency table analysis was used to interpret the data. Chi-square tests were performed, and odds ratios were used to estimate relative risk. Adjusted odds ratios were derived from logistic regression coefficients. Statistical significance was expressed in exact values. The criterion for significance was set at an alpha level of 0.05.

RESULTS

In total, we tested samples from 209 (93.3%) of 224 patients admitted to the unit for the homeless during the study period. Of the 209 samples sent for antibody testing, 19 were reactive on enzyme immunoassay. Of these 19 samples, six showed indeterminate results on the Western Blot. The Centers for Disease Control indicate that individuals with reported risk whose assays are indeterminate may be in the process of conversion of their HIV status but that most often, retesting results in repeated indeterminate Western Blots (22). We concluded that the HIV status of all patients with indeterminate results was equivocal and excluded data on

those six subjects, so that data on 203 patients were used in the analysis of relative risk and serostatus. The remaining 13 samples that were reactive on enzyme immunoassay were confirmed positive by Western Blot assay, for a seroprevalence of 13 of 203, or 6.4%. The distribution of positive results is shown in table 1. No sample that was reactive on the enzyme immunoassay was negative on the Western Blot assay.

Table 1 shows that the only significant demographic variable associated with seropositivity was age. Patients between the ages of 18 and 39 accounted for 51.2% (N=104) of the 203 subjects and 84.6% (N=11) of the 13 positive results, a seroprevalence of 10.6% for this subsample.

The estimates of relative risk presented in table 1 were derived by using unadjusted odds ratios to compare the risk of testing positive for HIV-1 antibodies for patients with and without identified risk factors. Unadjusted odds ratios estimate relative risk for each factor without controlling for effects of other risk variables. The unadjusted odds ratios show that patients under 40 years of age were nearly six times more likely to be seropositive than patients over 40 and that patients with recorded histories of injecting drugs were almost five times as likely to be seropositive as patients with no history of such drug use on their charts. Recorded homosexual activity among the men did not predict positive HIV status in this group of patients.

Multiple logistic regression was used to analyze simultaneously the effects of age and injecting drugs, which were significant in the univariate analyses. In ad-

dition, ethnicity, while not significantly related to seropositivity overall, was associated with age: more patients under 40 years of age than over 40 were black or Hispanic ($\chi^2=13.73$, $df=1$, $p=0.0002$). We therefore controlled for ethnicity in the regression analysis. The adjusted odds ratios in table 1 demonstrate that for patients who were less than 40 years old, the likelihood of testing positive for HIV-1 antibodies was more than six times greater, and having a recorded history of injecting drugs independently increased the risk of HIV seropositivity more than 6.5-fold.

When we examined the linked risk histories of the seropositive patients, we found that clinicians had identified risk behavior in three (23.1%) of the 13 patients. Among the 10 HIV-positive men, three (30%) engaged in known risk behaviors: one (10%) used injected drugs and engaged in homosexual activity, and two (20%) were users of injected drugs. None of the three HIV-positive women had a recorded history of injecting drugs. Of the 190 seronegative patients, 12 (6.3%) were identified as users of injected drugs. Among the 136 seronegative men, 10 (7.4%) had histories of homosexual activity. Men with chart histories of injected drug use were significantly more likely to be HIV positive than men without such histories ($\chi^2=4.01$, $df=1$, $p=0.05$).

DISCUSSION AND CONCLUSIONS

New York city and state governments created a single integrated program for persons who are gravely disabled, mentally ill, and living on the streets. This program provided us with a unique opportunity to conduct a seroprevalence study among a difficult-to-research group that has generated considerable public debate. While patients who could be discharged after acute care at Bellevue Hospital were not studied, most Homeless Emergency Liaison Project patients required extended care in the state hospital unit for the homeless at which we conducted this study. Our sample is therefore representative of a majority of patients removed from the streets of New York City and involuntarily hospitalized who require more intensive treatment and placement efforts. Moreover, we were able to study 93.3% of the eligible patients requiring this level of care.

The 6.4% HIV seroprevalence for these homeless patients admitted for extended care to a state hospital ward is not significantly different from the 5.5% HIV seroprevalence we previously reported for new admissions to similar state hospital wards serving predominantly domiciled patients (5). The prevalence of HIV among both the patients in the unit for the homeless and those in the general units exceeded the estimated rate of HIV infection in the general population in New York City for a corresponding time frame (5). In both groups schizophrenia was the most common diagnosis, and at least 75% of the patients had had two or more psychiatric admissions before the index hospitalization.

Women in both the unit for the homeless and the general units were as likely as men to be HIV positive. Sero-

positivity among domiciled and undomiciled mentally ill women (6.1% and 5.3%, respectively) was four to five times higher than that of women delivering babies in New York City during a comparable time frame (1.25%) (23). High-risk activity among women was inadequately identified in our studies, but previous reports suggest that heterosexual transmission may play an important role in HIV infection among severely mentally ill women (8).

We also found important differences between the patients in the unit for the homeless and those in the general units. Whereas age did not predict seropositivity in patients in the general acute care units, patients in the unit for the homeless who were 18–39 years of age were 6.5 times more likely to be infected than those aged 40–59. Among the general unit patients, black patients were six times more likely to be infected than nonblack patients. Among the homeless unit patients, ethnic differences were less pronounced. Although patients under 40 had the highest seropositivity and were more likely to be black or Hispanic than patients over 40, our relatively small sample, in which black and Hispanic subjects were underrepresented, did not yield any clear relation between ethnicity and seropositivity.

While the seroprevalence of subgroups of the severely mentally ill may not differ widely, predictors of infection clearly diverge. For example, in contrast to both of our public hospital samples, seropositive patients at a private hospital were disproportionately male (6), a finding that mirrors the pattern of infection in the general population in New York City, where men are six times more likely to be represented among AIDS cases than women (24). Variations in sociodemographic and clinical characteristics among inpatients in different settings may be associated, in turn, with differences in high-risk behavior and the likelihood of exposure to infected sexual or needle-sharing partners. It is therefore important for investigators to describe surveyed patient groups in detail and to use caution in generalizing seroprevalence findings from one group to another.

The patient group we studied was distinctive in many respects. These patients had been living on the streets, often for extended periods of time, and many avoided the public shelter system available to the homeless in New York City. Sixty-four percent of the patients treated during the first 6 months of the Homeless Emergency Liaison Project had lived primarily in public spaces and not in shelters before admission to Bellevue Hospital (E. Susser, personal communication, Oct. 16, 1991). Furthermore, we sampled only Homeless Emergency Liaison Project patients who were gravely disabled and required treatment at an extended care facility. They were forcibly transported to emergency rooms, involuntarily admitted to an acute care hospital, and subsequently transferred for extended care to a state hospital. The group we studied, therefore, is likely to have come from the most severely impaired subgroup of the homeless mentally ill, and generalizing these findings to better-functioning homeless individuals or those who require only a short hospital stay may not be appropriate.

The disadvantages of studying this special 70-bed unit for the homeless included the small number of new admissions during the study period, particularly among Hispanic patients. Moreover, because Hispanic patients were overrepresented among the 7% of patients from whom we could not obtain blood, the 15% HIV seropositivity for this ethnic group may be an artifact of undersampling. Previous studies have suggested that Hispanic persons are at greater risk for HIV infection than whites (25, 26), and special efforts should be made in future prevalence studies of the severely mentally ill to obtain a representative Hispanic sample, taking into account geographic region of origin, which is predictive of seropositivity (M.E. St. Louis et al., V International Conference on AIDS, 1989).

Other study limitations are intrinsic to the anonymous seroprevalence method (7). Among these is the need to restrict the number of variables linked to each blood sample to preserve patient anonymity. In addition, risk data were obtained from patient charts, because the anonymous study design did not permit interviews with patients. Only a minority of HIV-positive patients had risk histories on their charts, which limited our ability to make inferences about what behaviors were associated with HIV seropositivity, particularly among women. Moreover, since systematic risk assessments and psychiatric diagnoses could not be obtained by interview, we were unable to examine how psychiatric illness affects risk-taking behavior. Nevertheless, the anonymous method allowed us to obtain seroprevalence data on more than 200 street-dwelling homeless mentally ill individuals. These data represent the most complete epidemiological HIV profile of the homeless mentally ill to date and provide a basis for planning for the needs of this chronically underserved subgroup of the severely mentally ill.

Independent of the seroprevalence study, an in-depth chart review was conducted on the Creedmoor unit for the homeless that may suggest how age, ethnicity, and risk are related in this population. Compared to older patients, more of those under 40 were black or Hispanic and were more likely to use crack, a smokable analogue of cocaine (our unpublished data). Crack use is highly associated with HIV risk behavior (M.A. Chaisson et al., VI International Conference on AIDS, 1990). Crack-related high-risk behavior may account for the higher seroprevalence among patients under age 40 than among those 40 years of age or over.

Carefully conducted studies in multiple settings are needed to clarify the relation between age, ethnicity, and risk among the homeless mentally ill. Differences in psychopathology and lifestyle among subgroups of the homeless are also likely to play an important role. Street life may be associated with a pattern of drug use and sexual activity which is very different from that found in shelters for the homeless. As a result, different subgroups of the homeless may have a higher or lower prevalence of HIV infection than the group we studied.

We found that one in every 16 patients admitted to a special psychiatric unit for the homeless mentally ill was

HIV positive. Systematic procedures must be developed in psychiatric settings for inquiring about HIV-risk behavior. Aggressive investigation of risk histories is necessary for informing clinical decisions about testing and subsequent medical intervention and to stem transmission of HIV to uninfected patients. Fully 10% of the seronegative patients in our study had histories of high-risk activity on their charts. These patients should be targeted for risk reduction interventions to prevent their becoming seropositive.

Mentally ill people who live on the streets are unlikely to take advantage of traditional health care programs. Street-based interventions in which information on risk reduction and condoms are distributed have been effective with other disaffiliated populations (Y. Serrano and D. Goldsmith, V International Conference on AIDS, 1989). Similar outreach prevention efforts are urgently needed to reach the mentally ill living on the streets.

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Antisocial Personality Disorder and HIV Infection Among Intravenous Drug Abusers

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***Objective:** Antisocial personality disorder in drug abusers has been associated with poor treatment outcome. The authors examined the relationship between diagnosis of antisocial personality and HIV infection. **Method:** Subjects were 272 intravenous drug abusers, 140 (52%) of whom were in methadone treatment. Subjects were given an HIV risk behavior interview before diagnostic interviewing and HIV testing. **Results:** Using the DSM-III-R definition, the authors found that 119 (44%) of the subjects met criteria for antisocial personality. Significantly more of the subjects with antisocial personality (18% [N=21]) than of the subjects without antisocial personality (8% [N=12]) had HIV infection. The diagnosis of antisocial personality disorder was associated with a significantly higher odds ratio of infection independent of ethnicity, gender, and treatment status. **Conclusions:** Antisocial personality is a risk factor for HIV infection among intravenous drug abusers.*

(Am J Psychiatry 1993; 150:53-58)

Antisocial personality disorder is one of the most common psychiatric diagnoses among intravenous drug abusers. Using DSM-III criteria, Khantzian and Treece (1) and Rounsaville et al. (2) found rates of antisocial personality in drug abusers of 35% and 54%, respectively. More importantly, the co-occurrence of antisocial personality and intravenous drug abuse has been linked to poor drug abuse treatment outcome (3), even when specialized forms of psychotherapy were provided concurrent with routine counseling (4).

The poor treatment outcome of drug abusers with antisocial personality disorder is becoming increasingly important because continued intravenous drug use is strongly associated with risk of HIV infection (5, 6). Despite this, the relationship of antisocial personality to the risk of HIV infection has not been examined. Dolan et al. (7) investigated the relationships between MMPI scores and drug injection practices among 224 inpatient drug abusers. They found no statistically significant dif-

ferences in scores on the MMPI psychopathic deviate subscale (an index of psychopathy) between patients who never injected, those who injected but denied needle sharing, and those who injected and reported "selective or non-selective" needle sharing. This finding is not surprising because most drug abusers obtain high scores on the psychopathic deviate scale (8). Further, Hare (9) reported only a modest statistical association between psychopathic deviate scores and the diagnosis of antisocial personality.

Elsewhere (10), we examined the relationships between a diagnosis of antisocial personality disorder and high-risk drug use behaviors in 100 intravenous drug abusers. Drug abusers with the diagnosis of antisocial personality disorder reported significantly more needle sharing and a larger number of different needle-sharing partners than did subjects without the diagnosis. These findings suggest that the diagnosis of antisocial personality may identify a subgroup of intravenous drug abusers whose drug use behavior places them at higher risk of HIV transmission. Certainly, intravenous drug abusers are not homogeneous with respect to HIV risk. To date, higher HIV infection rates have been found for blacks and Hispanics, for males, and for those not enrolled in drug abuse treatment (5, 6, 11-14).

The present study reports on the relationship between a diagnosis of antisocial personality disorder and rate of HIV infection among intravenous drug abusers. The rates of HIV infection of subjects with and without antisocial personality were compared while other relevant variables such as ethnicity, gender, and treatment status were statistically controlled for. Additionally, drug in-

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jection and needle-sharing practices were examined in relation to diagnosis of antisocial personality disorder and to HIV infection.

METHOD

The 272 subjects were intravenous drug abusers enrolled in a longitudinal study of psychopathology, drug use practices, and HIV infection. The present data were all collected during the intake phase of that study. All subjects were of unknown HIV status at enrollment. Approximately half of the subjects were in methadone treatment ($N=140$) and were recruited by advertisements posted in drug treatment clinics and by chain-referral techniques. The remaining 132 subjects were not in methadone treatment and were recruited from advertisements in health and research centers and by chain-referral. These subjects reported using drugs intravenously within the 3 months before enrollment, had a history of at least 3 months of continuous intravenous drug abuse, and had received less than 12 cumulative months of methadone treatment in the previous 4 years.

Subjects were paid \$10.00/hour to participate in the confidential assessments, which took about 6 hours in all (\$60.00). Informed consent was obtained from all participating subjects after the study procedures were described. Ninety-four (35%) of the 272 subjects were in our previous study of antisocial personality and drug use patterns (10).

The mean age of the subjects was 36.3 years ($SD=7.1$), 196 (72%) were men, 163 (60%) were black, 103 (38%) were white, and six (2%) were Hispanic. The mean education level was 11.2 years ($SD=1.8$), 128 (47%) had never married, and 38 (14%) were married. The median cumulative lifetime duration of methadone treatment for the 140 patients who were receiving methadone was 36.5 months; for the 132 subjects who were not in methadone treatment the median was 0 months; for the total group of 272 subjects the median was 5 months. More of the subjects who were in methadone treatment were married (20% [$N=28$] versus 8% [$N=11$], $\chi^2=6.61$, $df=1$, $p<0.01$), and more of the subjects who were not receiving methadone were men (86% [$N=121$] versus 59% [$N=77$], $\chi^2=24.7$, $df=1$, $p<0.001$) and from an ethnic minority (89% [$N=118$] versus 36% [$N=51$], $\chi^2=78.77$, $df=1$, $p<0.001$).

Instruments

Information for making the DSM-III-R diagnoses of antisocial personality and substance use disorders was obtained from the Alcohol Research Center Intake Interview, a semistructured diagnostic interview with good reliability and procedural validity (15). The Alcohol Research Center Intake Interview was developed from items in the Schedule for Affective Disorders and Schizophrenia—Lifetime Version (16) and the National Institute of Mental Health Diagnostic Interview Sched-

ule (17). The interview covers lifetime and current diagnoses that have high prevalence among alcohol- and drug-dependent patients (e.g., antisocial personality disorder and major depression) and several categories (e.g., schizophrenia) with low prevalence but high morbidity. Modifications were made to the Alcohol Research Center Intake Interview to permit separate diagnoses of abuse or dependence for each of the major drug classes, excluding nicotine and caffeine.

Information on specific drug use behaviors with a high risk of HIV infection was obtained by conducting a structured interview assessing frequency of drug injections and needle-sharing episodes (i.e., giving or receiving previously used needles and syringes) and number of different needle-sharing partners (10). This incorporated a time-line follow-back approach: subjects initially provided detailed information on the frequency of drug injection and needle-sharing practices for each of the 7 days before study enrollment (summarized as week 1). The week-1 summary data were then used as a reference to collect an additional 3 weeks of information, summarized as month 1. The month-1 summary data were used as a reference to collect an additional 11 months of information, summarized as the 1-year data. Finally, the 1-year summary data were the reference for collecting an additional 4 years of information, summarized as the 5-year data. Although the reliability and validity of this procedure are undocumented, it was considered preferable to obtaining estimates of these past behaviors without recent reference values.

Data on number of drug injections, needle sharing, and number of different needle-sharing partners were tabulated for each of the 12 months to summarize the past year of drug use behavior and for each of the 5 years to obtain the 5-year average. Subjects were also asked if they had shared needles with the following people: 1) someone who later died of an AIDS-related illness, 2) someone with AIDS, 3) someone believed to have AIDS, 4) someone with HIV infection, and 5) someone believed to be HIV infected.

Procedure

Subjects completed the assessments in three sessions. The HIV risk behavior interview preceded the Alcohol Research Center Intake Interview, and both of these interviews preceded the HIV informing session. Assessments were conducted by four master's-level trained technicians. Each completed 4 hours of Alcohol Research Center Intake Interview didactic training, co-rated a minimum of five consecutive interviews with agreement on diagnoses made by an expert interviewer, and conducted five consecutive interviews that agreed with diagnoses made by expert co-raters.

Blood samples for HIV testing were analyzed by enzyme-linked immunosorbent assay (ELISA) and confirmed by the Western Blot (Dupont Kit). Subjects were informed of their HIV test result by confidential interview, and cases of infection were referred to our AIDS service for further evaluation and care.

Statistical Analyses

Comparisons of group differences were made by using chi-square with Yates's correction for categorical variables (or Fisher's exact test for comparisons involving small cell sizes), Student's *t* test for means, and the Mann Whitney U test for medians (transformed to a *z* statistic for large numbers of subjects). The univariate relationships of antisocial personality disorder, demographics, and treatment status with HIV infection were examined in two-by-two tables. Odds ratios for infection were calculated and tested for significance by using Fisher's exact test. The multivariate association of antisocial personality disorder with HIV infection was examined in two-by-two tables stratified by minority and nonminority, male and female, and methadone treatment and no methadone treatment. Statistical methods described by Rothman (18) and a computer program developed by Foster and Sullivan (19) were used. Maximum likelihood estimation was used in these stratification analyses; likelihood ratio chi-squares (*df*=1) tested the hypotheses that the association of antisocial personality with HIV infection was uniform across strata (i.e., meaning no interaction). Given the absence of significant interaction, maximum likelihood estimation provided the uniform odds ratios and 95% confidence intervals for infection in subjects with versus subjects without antisocial personality. The significance of the odds ratios was tested by using an exact test for stratified case-control data (related to Fisher's exact test). A similar analysis simultaneously stratified by minority status, gender, and current methadone treatment status.

For analyses of number of injections and needle-sharing occasions, comparisons were made for the total of the 12 preceding months and for the average of years 1–5 (5-year average). Medians rather than means were compared because these distributions were highly skewed. Similarly, the distribution for number of different needle-sharing partners was skewed for each time period (i.e., 1 year and the 5-year average). To examine the relationships of multiple needle-sharing partners and infection, subjects were divided into those who reported more than one partner and those with one or no partners for each time period; this split yielded enough subjects in each group for statistical comparison.

RESULTS

Prevalence and Correlates of Antisocial Personality Disorder

Of the 272 subjects, 119 (44%) met DSM-III-R criteria for antisocial personality disorder. More of the antisocial subjects were men (86% [*N*=102] versus 61% [*N*=94], $\chi^2=18.40$, *df*=1, *p*<0.001) and fewer were in methadone treatment at study entry (44% [*N*=52] versus 58% [*N*=88], $\chi^2=4.58$, *df*=1, *p*<0.03). There were no significant differences between subjects with and without antisocial personality in age (mean=36.6 versus mean=36.1, *t*=0.65, *df*=62, *p*=0.51), ethnicity (66% [*N*=79] versus 59%

[*N*=90] nonwhite, $\chi^2=1.32$, *df*=1, *p*=0.25), or years of education (11.0 versus 11.4, *t*=1.77, *df*=70, *p*=0.09).

The rates for several lifetime drug use diagnoses (abuse and dependence combined) were higher for subjects with antisocial personality than for those without, including cocaine (88% [*N*=105] versus 76% [*N*=116], $\chi^2=6.11$, *df*=1, *p*<0.01), alcohol (77% [*N*=92] versus 45% [*N*=68], $\chi^2=30.07$, *df*=1, *p*<0.0001), sedatives (50% [*N*=59] versus 34% [*N*=52], $\chi^2=5.90$, *df*=1, *p*<0.02), and hallucinogens (17% [*N*=20] versus 7% [*N*=11], $\chi^2=5.12$, *df*=1, *p*<0.02). No significant group differences were found for opioids (92% [*N*=110] versus 92% [*N*=141], $\chi^2=0.01$, *df*=1), marijuana (55% [*N*=66] versus 46% [*N*=70], $\chi^2=2.02$, *df*=1), amphetamines (14% [*N*=17] versus 9% [*N*=14], $\chi^2=1.23$, *df*=1), or inhalants (5% [*N*=6] versus 1% [*N*=1], $\chi^2=3.50$, *df*=1). The diagnosis of antisocial personality was also associated with having a greater number of drug use disorders (3.98 versus 3.09, *t*=5.67, *df*=269, *p*<0.0001).

Risk Factors Associated With HIV Infection

Thirty-three (12%) of the 272 subjects were HIV positive. Table 1 shows the rates and odds ratios of HIV infection and the significance levels for each of the five variables under investigation. Having a diagnosis of antisocial personality disorder, minority ethnicity, lack of current methadone treatment, and a smaller cumulative lifetime duration of methadone treatment were each significantly associated with a higher rate of HIV infection.

The results of the stratification analyses examining the relationships between diagnosis of antisocial disorder and HIV infection when the effects of ethnicity, gender, and current methadone treatment status were statistically controlled for are shown in table 2. Current methadone treatment status was chosen for inclusion in these analyses because it was univariately more strongly related to infection than cumulative lifetime duration of methadone treatment. After controlling individually for ethnicity, gender, and current methadone treatment, antisocial disorder was in each case significantly associated with a greater overall risk of HIV infection. For example, 25.3% of the minority subjects with antisocial personality disorder versus 13.3% of those without the diagnosis were infected, and 2.5% of the nonminority subjects with antisocial personality versus none of those without the diagnosis were infected.

After ethnicity, gender, and current methadone treatment status were simultaneously controlled for, the hypothesis of a uniform odds ratio was confirmed ($\chi^2=5.02$, *df*=7, *p*=0.66) and the diagnosis of antisocial personality disorder was associated with a significantly higher infection rate (uniform odds ratio=2.44, 95% confidence interval=1.06–5.49, *p*=0.04 [exact test]).

Antisocial Personality Disorder and HIV-High-Risk Behaviors

Significant differences were found between the subjects with and those without antisocial personality in

TABLE 1. Demographic and Clinical Characteristics and HIV Infection Rates of Intravenous Drug Users With and Without Antisocial Personality Disorder

Characteristic	Number of Subjects	Subjects With HIV Infection		Odds Ratio	95% Confidence Interval	p ^a
		N	%			
Diagnosis				2.51	1.18–5.49	0.02
Antisocial personality disorder	119	21	17.6			
No antisocial personality disorder	153	12	7.8			
Gender				1.86	0.76–5.14	0.19
Men	196	27	13.8			
Women	76	6	7.9			
Ethnicity				23.82	4.39–494.87	0.001
Minority	169	32	18.9			
Nonminority	103	1	1.0			
Current methadone treatment				3.23	1.46–7.58	0.004
No	132	24	18.2			
Yes	140	9	6.4			
Lifetime duration of methadone treatment				2.60	1.20–5.93	0.02
0–4 months	135	23	17.0			
4 or more months	137	10	7.3			

^aFisher's exact test.**TABLE 2. HIV Infection in Intravenous Drug Users With and Without Antisocial Personality Disorder With Other Variables Controlled**

Variable Controlled	Subjects With Antisocial Personality			Subjects Without Antisocial Personality			Uniform Odds Ratio	95% Confidence Interval	p ^a
	Total	Subjects With HIV Infection		Total	Subjects With HIV Infection				
		N	%		N	%			
Ethnicity							2.35	1.07–5.25	0.03
Minority	79	20	25.3	90	12	13.3			
Nonminority	70	1	2.5	63	0	0.0			
Gender							2.32	1.06–5.17	0.04
Men	102	19	18.6	94	8	8.5			
Women	77	2	11.8	59	4	6.8			
Current metha- done treatment							2.23	1.04–4.92	0.04
No	67	15	22.4	65	9	13.8			
Yes	52	6	11.5	88	3	3.4			

^aThe significance of the odds ratios for infection in those with versus those without antisocial personality was tested by using an exact test for case-control data. In each case the test for uniformity of the odds ratios across strata indicated no significant interaction (for ethnicity, $\chi^2=1.06$, $df=1$, $p=0.30$; for gender, $\chi^2=0.09$, $df=1$, $p=0.77$; for treatment, $\chi^2=0.72$, $df=1$, $p=0.40$).

the median number of drug injections, the median number of needle-sharing occasions, and the percentage reporting multiple needle-sharing partners for the past year and the 5-year average (figures 1 and 2). In all cases the direction of the difference was toward more risk of HIV exposure in those with antisocial personality.

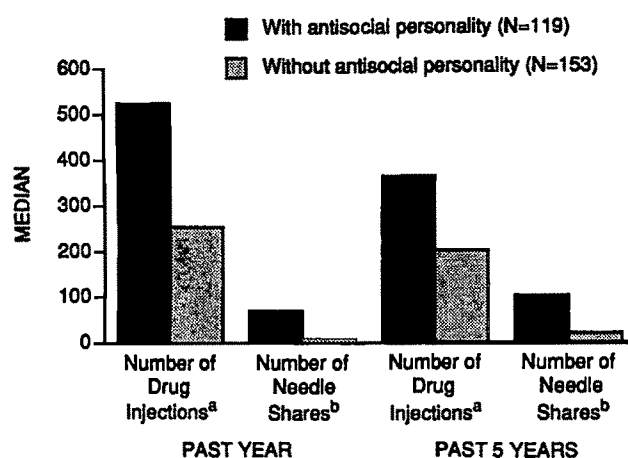
Medians are reported because the distributions of scores were extremely skewed. In year 1, the lowest 10% of the group of 272 subjects reported 0–3 injections and the highest 10% reported more than 1,355 injections. Similarly, 79 (29%) of the subjects reported no needle sharing in the first year, but 14 (5%) reported needle sharing on more than 970 occasions with over 100 different people. For both the past year and the 5-year average, the subjects with antisocial personality disorder reported significantly more drug injections and needle sharing than subjects without the diagnosis, and a greater proportion of the subjects with antisocial

disorder also had multiple needle-sharing partners. More of the subjects with antisocial personality also shared needles with someone who later died of an AIDS-related illness (15% [N=18] versus 7% [N=10], $\chi^2=4.57$, $df=1$, $p<0.03$).

High-Risk Drug Use Behaviors and HIV Infection

Only one of the nine high-risk drug use behaviors examined was significantly associated with HIV infection. Specifically, HIV-infected subjects reported a higher average number of yearly drug injections for the previous 5 years than did those who were not infected (median=530.4 versus median=235.2, $z=1.61$, $p<0.05$); this analysis was nonsignificant when the p value was adjusted for multiple comparisons. The eight remaining variables for the HIV-infected versus noninfected subjects included number of injections in year 1 (median=526.0 versus median=336.5, $z=1.61$, n.s.), number of

FIGURE 1. Number of Drug Injections and Needle-Sharing Occasions of Intravenous Drug Users With and Without Antisocial Personality Disorder



^aAntisocial personality was associated with more drug injections in the past year ($z=2.83$, $p<0.005$, Mann-Whitney U test) and in the past 5 years ($z=3.31$, $p<0.001$, Mann-Whitney U test).

^bAntisocial personality was associated with more needle-sharing occasions in the past year ($z=4.35$, $p<0.00001$, Mann-Whitney U test) and in the past 5 years ($z=3.82$, $p<0.0001$, Mann-Whitney U test).

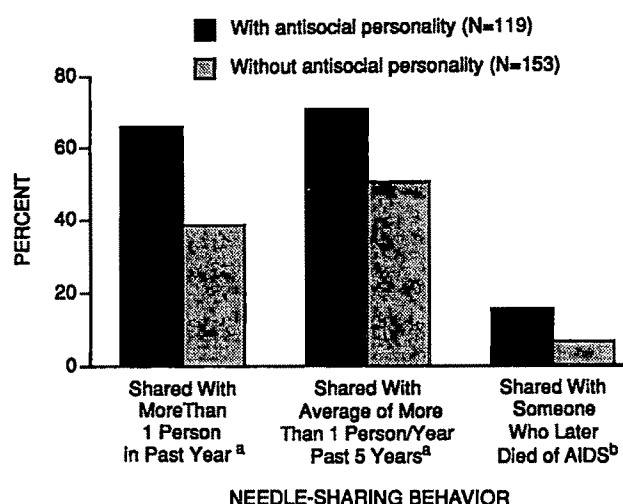
needle-sharing occasions in year 1 (median=46 versus median=15, $z=0.72$, n.s.), average number of needle-sharing occasions in years 1–5 (median=43.2 versus median=39.6, $z=0.34$, n.s.); percent sharing needles with more than one person in year 1 (52% [N=17] versus 50% [N=120], $\chi^2=0.002$, $df=1$, n.s.); percent sharing needles with more than one person/year in years 1–5 (67% [N=22] versus 58% [N=139], $\chi^2=0.55$, $df=1$, n.s.); and percent sharing needles with persons who later died of an AIDS-related illness (21% [N=7] versus 9% [N=21], n.s. [Fisher's exact test]).

DISCUSSION

The positive relationship between antisocial personality disorder and HIV infection indicates an important biobehavioral association. This association between psychiatric diagnosis and infection status is especially remarkable given that it was observed among intravenous drug abusers, a group known to have a high level of HIV risk and risk behaviors. Importantly, the higher infection rate among drug abusers with antisocial personality disorder was not due to ethnicity, gender, or drug treatment factors previously linked to higher infection rates in this population.

The association of antisocial personality with HIV infection presumably occurs through behavioral mechanisms. Although these mechanisms could not be determined from the present data, the diagnosis of antisocial personality disorder is characterized by chronic irresponsible, exploitative, and reckless behaviors in the context of poor impulse control and low harm avoidance. It is reasonable to surmise that these behavioral

FIGURE 2. Intravenous Drug Users With and Without Antisocial Personality Disorder Who Shared Needles With More Than One Person or Who Shared Needles With Someone Who Later Died of an AIDS-Related Illness



^aMore subjects with antisocial personality reported having shared needles with more than one person in the past year ($\chi^2=18.62$, $df=1$, $p<0.00002$), and more subjects with antisocial personality reported sharing needles with an average of more than one person per year for the past 5 years ($\chi^2=10.55$, $df=1$, $p<0.008$).

^bMore subjects with antisocial personality reported sharing needles with someone who subsequently died of AIDS-related illness ($\chi^2=4.57$, $df=1$, $p<0.03$).

characteristics associated with antisocial personality may contribute to a greater risk of HIV infection. In the present study, high-risk drug use behaviors were generally more pervasive and more frequent among those with than among those without antisocial personality disorder. These findings generally replicate those obtained in our previous study of 100 subjects (10). The one exception was that the earlier study did not find a significant association between antisocial personality and number of drug injections; this difference may be due to the greater statistical power of the present study.

It is also possible that greater risk behavior and HIV infection among drug abusers with antisocial personality disorder may relate to their having a more severe spectrum of drug use disorder (7). Some evidence for this was found in the present study. Antisocial drug abusers had significantly higher rates of cocaine, alcohol, sedative, and hallucinogen abuse or dependence, as well as a greater number of lifetime diagnoses, than did the subjects without antisocial personality. This drug abuse pattern could have further reduced impulse control for subjects with antisocial personality disorder and contributed to greater risk behavior (e.g., injections and needle sharing) and HIV infection, particularly given their high rates of cocaine abuse (11, 14).

The lack of strong relationships between HIV infection and specific patterns of intravenous drug use was somewhat surprising. This may have occurred because intravenous drug use and needle sharing were common behaviors in the population and the infection rate was

relatively low (12%). This combination of factors could obscure the relationship between needle sharing and infection (6). The lack of more consistency between our findings and earlier reports of positive associations between needle sharing and HIV infection could have resulted from differences in risk assessment instruments and procedures (5, 12, 13). Alternatively, it is possible that although indexes of intravenous drug use are important predictors of HIV infection risk in the general population (where such drug use is relatively rare), they may be less stable predictors of infection in populations where intravenous drug use and needle sharing are commonplace.

Because the sexual practices of subjects were not examined, relationships between antisocial personality, high-risk sexual behaviors, and HIV infection could not be determined. Another potential study limitation is that our subjects may not be representative of other intravenous drug abusers. Finally, caution is necessary in extrapolating our findings to white subjects or non-black minority populations because most of the cases of infection we found were among black subjects.

In conclusion, these data emphasize the importance of antisocial personality in the assessment of risk of HIV infection among drug abusers. Although intravenous drug abusers are at greater behavioral risk of HIV infection than the general population, they are not homogeneous in this regard. It is both possible and important to identify differences among subjects that are significantly related to the risk of infection; the diagnosis of antisocial personality appears to be one such factor. The data also add new importance to the treatment of intravenous drug abusers with antisocial personality disorder. Although little is known about factors that may improve the treatment outcome of this group, work by Woody et al. (4) suggests that intensified treatment of drug abusers with antisocial personality disorder is associated with reduced drug use. Even modest reductions in the drug use of antisocial patients may translate into reduced risk of HIV transmission.

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Structural Abnormalities in Deficit and Nondeficit Schizophrenia

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***Objective:** Previous studies have suggested the involvement of the frontal and parietal cortices and thalamus in a neural circuit underlying the production of primary enduring negative or deficit symptoms of schizophrenia. The purpose of this study was to examine whether structural changes in the proposed circuit are associated with the production of deficit symptoms. **Method:** Magnetic resonance imaging was used to measure the volume of selected circuit brain regions (i.e., the prefrontal region and caudate) and noncircuit brain regions (i.e., the amygdala/hippocampus complex) in 17 deficit and 24 nondeficit schizophrenic outpatients and 30 normal comparison subjects. **Results:** Right and left total prefrontal volumes discriminated deficit from nondeficit patients, with prefrontal volumes being smaller in nondeficit patients. There were no differences between the two schizophrenic subgroups in left caudate or right and left amygdala/hippocampus complex volumes. The right caudate was larger in deficit patients, but the difference between the two schizophrenic subgroups was not significant. There were no differences between deficit and normal subjects on any prefrontal region measure. Nondeficit patients had smaller total right and left prefrontal volumes than normal subjects. Both schizophrenic subgroups had larger left caudate volumes and smaller right and left amygdala/hippocampus complex volumes than the normal subjects. There was a trend for deficit patients to have larger right caudate volumes. **Conclusions:** These results suggest that structural changes in the prefrontal region are not responsible for deficit symptoms. The caudate, particularly the right caudate, may be associated with the production of these symptoms. (Am J Psychiatry 1993; 150:59-65)*

There is considerable interest in delineating which areas of the brain are involved in the production of negative symptoms in schizophrenia (1). The most commonly reported finding, in previous studies using computed tomography (CT) and magnetic resonance imaging (MRI), has been the greater number of negative symptoms in patients with enlarged lateral ventricles (2-7). However, a number of studies have failed to find a significant relation between ventricular enlargement and negative symptoms (8-15), and at least two studies have found the opposite relationship, i.e., more negative symptoms in subjects with smaller ventricles (8, 16).

Several lines of evidence have implicated the fron-

tal—especially the prefrontal—cortex in the production of negative symptoms (17-19). Five imaging studies have examined the relation of negative symptoms to morphological characteristics of the frontal lobe. Ota et al. (12) found a trend for negative symptoms to be associated with prefrontal lobe atrophy. Andreasen and co-workers (7, 20) conducted two MRI studies examining the relation between frontal lobe, cerebral, and cranial area measurements and negative symptoms. The two studies differed in the criteria used to select the normal control subjects, with emphasis placed in the second study on selecting normal control subjects with a level of education equivalent to that of the schizophrenic patients. In the first study, these investigators observed that patients with smaller cerebral and cranial area measurements had significantly more negative symptoms (20). However, they were not able to document, in either study, a relation between negative symptoms and the midsagittal frontal lobe area (7, 20). Data on the relation between cerebral and cranial area measurements and negative symptoms were not reported in

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the second study. Besson et al. (21) reported greater regional spin-lattice relaxation time (T_1) (a measure thought to be sensitive to the presence of pathological processes) in the left prefrontal white matter of patients with high levels of negative symptoms than of patients with low levels. In contrast to the study of Ota et al. (12), Pfefferbaum et al. (14) found negative symptoms to be inversely correlated with frontal sulcal size.

These studies were unable to demonstrate a consistent association between structural abnormalities and negative symptoms. If negative symptoms have a true association with altered brain structure, the inconsistent findings may be due to 1) the marked variability between studies in the assessment of negative symptoms, 2) the failure to distinguish primary and enduring negative symptoms from transitory and secondary negative symptoms (22, 23), and 3) limitations of the imaging methodologies used in these studies. CT scans do not allow for precise differentiation of gray matter from white matter, and in the MRI studies, measurements of area rather than volume were used.

An alternative approach for studying the pathophysiology of negative symptoms is to restrict the focus of study to primary, enduring negative or deficit symptoms and to define putative subgroups of schizophrenic patients on the basis of the presence or absence of these symptoms (24, 25). Validation of this approach has been provided by comparing patients with and without deficit symptoms on a number of clinical and functional measures hypothesized to be related to deficit symptoms (26–29). In addition to demonstrating that deficit patients have poorer premorbid adjustment and greater social and physical anhedonia (26, 29), these studies have shown that patients with deficit symptoms 1) are more neurologically impaired (29), especially on items sensitive to parietal lobe function; 2) exhibit increased volitional saccadic latency (27), an eye-tracking measure sensitive to frontal and/or parietal lobe impairment (30); and 3) have reduced glucose utilization, as assessed with positron emission tomography (PET), in the frontal and parietal cortices and thalamus (28).

A model for integrating these results is provided by the work of Alexander et al. on basal ganglia-thalamo-cortical circuits (31, 32). They have proposed the existence of parallel segregated circuits, each of which is hypothesized to subserve a discrete range of functions. Each of these circuits is characterized by cortical projections to the striatum, projections from the striatum to the substantia nigra/globus pallidus, projections from the substantia nigra/globus pallidus to the thalamus, and, to complete the circuit, projections from the thalamus to the frontal cortex (32). One of these circuits, the dorsolateral prefrontal circuit, is of particular relevance, as it is composed of the brain regions (i.e., the dorsolateral prefrontal and posterior parietal cortices and thalamus) implicated in the pathophysiology of the deficit syndrome (27–29).

To investigate the involvement of the hypothesized neural circuit in the production of deficit symptoms, a convergence-of-evidence approach is chosen, whereby

multiple measures (e.g., PET and neurobehavioral assessments) are used to assess the functional integrity of the circuit, and MRI and post-mortem assessments are used to evaluate the structural integrity of the hypothesized circuit. In the present study, to test the hypothesis that structural changes in components of the dorsolateral prefrontal circuit are associated with deficit symptoms, we used MRI to measure the volumes of selected components of the proposed circuit (i.e., the prefrontal region and caudate) and of a brain region outside the proposed circuit (i.e., the amygdala/hippocampus complex) in schizophrenic patients with and without deficit symptoms. The results from the schizophrenic subgroups were compared to similar measures obtained from normal comparison subjects. The comparison between the total schizophrenic group and the normal subjects has been presented elsewhere (33).

METHOD

Forty-one patients with schizophrenia diagnosed according to the DSM-III-R criteria were selected from an outpatient research clinic for entry into the study. All gave informed consent. Diagnosis of the patients was done with a best-estimate approach that used all available sources of information, including direct assessment, family informants, and past medical records. Patients with organic brain disorders, mental retardation, histories of severe head trauma, and histories of drug abuse, dependence or alcoholism were excluded from the study. The patients were subtyped into deficit ($N=17$) and nondeficit ($N=24$) subgroups on the basis of the consensus diagnosis of two psychiatrists (R.W.B. and B.K.), using the Schedule for the Deficit Syndrome (34), a semistructured interview of documented reliability. The Schedule for the Deficit Syndrome provides specific criteria for assessing the presence of negative symptoms, the duration of the symptoms, and whether the symptoms are primary or secondary. All patients were interviewed while they were between episodes, during clinical stability. Additional information, if needed, was obtained from clinicians who had had long-standing contact with the patients and from family members.

The deficit subgroup consisted of 12 male and five female patients; eight were black and nine were white. Thirteen were right-handed and four were left-handed. The nondeficit subgroup contained 14 male and 10 female patients; seven were black and 17 were white. Nineteen were right-handed and five were left-handed.

To provide a framework for interpreting the results of the volumetric comparisons between the deficit and nondeficit patients, the same volumetric measures were obtained from 30 normal comparison subjects selected from the general population. The normal subjects had no past or current DSM-III-R axis I or axis II disorder as determined by the Structured Clinical Interview for DSM-III-R—Patient Version (35) and the Structured Interview for DSM-III Personality Disorders, 2nd ed. (unpublished manual by B. Pfohl et al.) and did not

have histories of organic brain disorder, mental retardation, or severe head trauma. This group consisted of 20 male and 10 female subjects; four were black, 25 were white, and one was "other." Twenty-five were right-handed and five were left-handed.

The MRI studies were performed on a Siemens 2-tesla Magnetom system operating at 1.5 teslas. Subjects' heads were positioned in a head coil fixation device centered at the orbitomeatal line with no angulation. A sagittal scout image was first acquired to correct obvious head tilt and to localize imaging coordinates. The high-resolution spin-echo technique was used to evaluate the whole brain in the coronal plane in 3-mm-thick contiguous slices, with a repetition time of 600 msec, an echo time of 17 msec, and a matrix size of 256×256 pixels, with two excitations. The MRI data, obtained on magnetic tape, were directly transferred to optical disk for archival purposes.

Morphometric analyses were performed with the Loats image analysis system (36). The sample function of the image analysis system was used to determine the caudate and amygdala/hippocampus complex volumes. This function enables the investigator to outline the region of interest directly on the MRI image and calculates the area of the demarcated region. Volumes are calculated by summing the area measurements across all appropriate images and multiplying by the slice thickness. The threshold function of the image analysis system was used to determine the volumes of the prefrontal gray and white matter. This function enables the investigator to partition gray matter from both cerebrospinal fluid (CSF) and white matter by assigning nonoverlapping signal intensity ranges to each area (36). CSF and gray and white matter were partitioned on each MRI slice containing the prefrontal region, and the sample function was used to outline the prefrontal hemispheres, generating separate gray and white matter area measurements. Volumes were calculated by summing the area measurements and multiplying by the slice thickness. The validity of the threshold procedure used in this study was supported by the significant negative correlations between right and left prefrontal gray matter volumes and age for the entire study group ($N=68$; $r=-0.32$, $p=0.007$, and $r=-0.33$, $p=0.006$, respectively) and the schizophrenic patients when considered alone ($N=39$; $r=-0.39$, $p=0.015$, and $r=-0.43$, $p=0.007$, respectively) (37, 38).

Although the thalamus is a part of the proposed circuit, thalamic volume was not measured because the poor separation of the thalamus from adjacent white matter on T_1 -weighted coronal images makes the reliable delineation of its boundaries difficult.

A system of rules based on brain atlases (39, 40) and published MRI studies (41–43) was used to generate landmarks for delineating the boundaries of the regions of interest. These landmarks served as general guidelines to supplement the information derived from visual inspection of the MRI images.

The anterior boundary of the prefrontal region was the first anterior coronal slice containing gray matter.

The posterior boundary was defined by the first slice demonstrating the genu of the corpus callosum. These landmarks for the prefrontal region were chosen because of their use in previous MRI studies (41–43). The anterior boundary of the caudate was the first slice containing the caudate, and the posterior boundary was the last slice containing the caudate before it turns laterally and caudally. The anterior slice contained the genu of the corpus callosum and the anterior horns of the lateral ventricles, and the posterior slice was at the level of the trigone of the lateral ventricle and the splenium of the corpus callosum. The tail of the caudate was not measured. The anterior anatomical landmark for the amygdala/hippocampus complex was the first slice containing the amygdala. This was usually the first slice containing the optic tracts and/or chiasm and the temporal horns of the lateral ventricle. The posterior boundary of the complex was defined as the most posterior slice containing the hippocampus. This slice was typically at the level of the splenium of the corpus callosum and the trigone of the lateral ventricle. The volumes of the amygdala and hippocampus were combined, since it was difficult to separate the anterior portion of the hippocampus from the posterior portion of the amygdala (44, 45).

Total cranial volume was determined for each subject to control for group differences in region-of-interest measurements due to differences in head size. Total cranial volume was defined as the total volume of cortical and subcortical gray and white matter plus total sulcal and ventricular CSF volume. The cerebellum and brainstem were not included in the determination of total cranial volume. Total cranial volume was determined by using the sample function to outline the edge of the dura mater on every other MRI image and multiplying the sum of the area measurements by the distance between the slices (6 mm).

The intraclass correlation coefficients for these three areas and total cranial volume ranged from 0.90 to 0.99. All quantifications were performed blind to any identifying information. The prefrontal volumes of two schizophrenic patients and one normal comparison subject and the caudate volumes of three schizophrenic patients could not be determined because of movement artifact. The total cranial volume of one normal subject could not be determined because a complete set of coronal images was not obtained.

Analyses of covariance, with age and gender as covariates, were performed to make comparisons of the brain region volumes between the schizophrenic deficit and nondeficit subgroups and between the schizophrenic subgroups and the normal subjects. Age and gender were used as covariates to control for the potential confounding effect of these variables on brain morphology (37, 38, 46, 47). Analyses of variance and chi-square tests were used where appropriate to compare total cranial volume, demographic variables, and clinical variables between the deficit and nondeficit subgroups and between the schizophrenic subgroups and the normal subjects.

TABLE 1. Clinical and Demographic Characteristics of Deficit and Nondeficit Schizophrenic Patients and Normal Comparison Subjects

Variable	Schizophrenic Patients				Normal Comparison Subjects	
	Deficit (N=17)		Nondeficit (N=24)		(N=20)	
	Mean	SD	Mean	SD	Mean	SD
Age (years)	35.5	6.6	35.6	6.0	34.0	8.1
Education (years)	11.6	1.4	13.2	2.4	14.3	2.2
Socioeconomic status ^a	4.7	0.6	3.6	1.0	3.1	0.7
Socioeconomic status of head of household ^a	3.7 ^b	0.5	2.8 ^c	1.2	3.1	1.2
Height (inches)	68.1	4.0	67.3	4.0	68.3 ^d	3.9
Age at onset (years)	20.0	6.8	22.2	5.7		
Duration of illness (years)	15.3	6.5	13.4	6.0		

^aRated from 1 to 5; higher numbers represent poorer status.^bN=14.^cN=21.^dN=29.

RESULTS

The deficit and nondeficit schizophrenic patients were similar with respect to age, sex, race, age at onset of psychosis, and duration of illness (table 1). The deficit patients had significantly fewer years of education ($F=5.99$, $df=1$, 39, $p<0.02$) and lower socioeconomic status ($F=15.55$, $df=1$, 39, $p<0.0001$). The socioeconomic status of the head of household of the family of origin was also lower for the deficit patients ($F=8.20$, $df=1$, 33, $p<0.01$).

The volumes of the right and left prefrontal region, caudate, and amygdala/hippocampus complex for the deficit and nondeficit patients and normal comparison subjects are presented in table 2. Deficit patients in comparison to nondeficit patients, had significantly larger total right prefrontal volume ($F=5.68$, $df=1$, 35, $p=0.02$) and total left prefrontal volume ($F=5.28$, $df=1$, 35, $p=0.03$), significantly larger right prefrontal white matter volume ($F=4.86$, $df=1$, 35, $p=0.03$), and a trend for larger left prefrontal white matter volume ($F=3.58$, $df=1$, 35, $p=0.07$). There were no statistically significant differences between the two schizophrenic subgroups in prefrontal gray matter volume. There also were no significant differences between these subgroups in the total volumes of the right and left caudate and the amygdala/hippocampus complex.

There were no differences between the deficit patients and the normal comparison subjects on any prefrontal measure. The deficit patients had significantly larger left caudate volumes ($F=7.12$, $df=1$, 43, $p=0.01$) and a trend toward larger right caudate volumes ($F=2.76$, $df=1$, 43, $p=0.10$) than the normal subjects. Deficit patients had significantly smaller right ($F=4.39$, $df=1$, 43, $p=0.03$) and left ($F=7.20$, $df=1$, 43, $p=0.01$) amygdala/hippocampus complex volumes than the normal subjects.

Nondeficit patients, as compared to normal comparison subjects, had smaller total right and left prefrontal volumes ($F=17.63$, $df=1$, 47, $p<0.0001$, and $F=8.31$, $df=1$, 43, $p<0.01$, respectively) and smaller right and left prefrontal white matter volumes ($F=13.55$, $df=1$, 43, $p=0.001$, and $F=7.17$, $df=1$, 43, $p=0.01$, respectively). There was a trend for the nondeficit patients to have smaller right prefrontal gray matter ($F=3.23$, $df=1$, 43, $p=0.08$). There was no difference between nondeficit patients and normal comparison subjects in right prefrontal gray matter. The left caudate was significantly larger in nondeficit patients ($F=4.67$, $df=1$, 47, $p=0.04$). There were no differences between the two groups in right caudate volume. There were significant differences between nondeficit patients and normal subjects in right and left amygdala/hippocampus complex volumes ($F=11.35$, $df=1$, 50, $p=0.001$, and $F=11.03$, $df=1$, 50, $p=0.002$, respectively), with the amygdala/hippocampus complex volumes being smaller in the nondeficit patients.

The deficit and nondeficit patients and the normal subjects were compared on total cranial volume to determine whether group differences in the volumetric measures were due to differences in total cranial volume. There was no difference in total cranial volume between deficit patients and nondeficit patients ($F=0.25$, $df=1$, 37, $p=0.62$), deficit patients and normal subjects ($F=0.03$, $df=1$, 42, $p=0.86$), and nondeficit patients and normal subjects ($F=0.78$, $df=1$, 49, $p=0.38$).

Secondary analyses were performed on all volumetric data to evaluate what effect, if any, the observed group differences in the socioeconomic status of the head of household had on the results from the primary analyses. Age and gender continued to be used as covariates in the analyses. The addition of household socioeconomic status as a covariate did not significantly alter the pattern of results from the primary analyses, although the differences between deficit and nondeficit patients in right and left total prefrontal volumes were reduced to trend levels. However, the effect size was the same as in the primary analyses. The decrease in F value was most likely due to the decrease in sample size from 39 to 34, because of missing data on household socioeconomic status, and the subsequent loss of statistical power. Although there were no differences in total cranial volume among the schizophrenic subgroups and the normal comparison subjects, the analyses were also done with total cranial volume added as a covariate. This did not alter the results of the primary analyses.

To examine possible differences between the groups in right/left asymmetry in prefrontal, caudate, or amygdala/hippocampus complex volumes, multivariate analyses of covariance were performed, with age and gender as covariates and side (right/left) as the within-subjects factor. The caudate was the only structure for which there was a significant Diagnosis by Side interaction. The interaction was significant in the deficit/nondeficit patients comparison ($F=4.65$, $df=1$, 34, $p<0.04$) and the nondeficit patient/normal subject comparison ($F=21.27$, $df=1$, 47, $p<0.001$) but not in the

TABLE 2. Prefrontal, Caudate, Amygdala/Hippocampus, and Total Cranial Volumes^a of Deficit and Nondeficit Schizophrenic Patients and Normal Comparison Subjects

Variable	Schizophrenic Patients				Normal Comparison Subjects (N=30) ^c	
	Deficit (N=17)		Nondeficit (N=24) ^b		Mean	SD
	Mean	SD	Mean	SD		
Prefrontal region						
Total volume						
Right	84.2	17.9	74.9	9.6	85.5	9.1
Left	80.8	14.5	73.5	10.0	81.2	9.0
Gray matter						
Right	49.0	10.2	45.2	6.5	48.3	5.6
Left	41.4	9.2	38.4	7.3	40.0	5.6
White matter						
Right	35.1	9.9	29.8	6.6	37.2	7.2
Left	39.3	8.6	35.1	7.5	41.2	7.9
Caudate, total volume						
Right	5.05	0.92	4.57	0.73	4.67	0.80
Left	4.62	0.83	4.41	0.58	4.06	0.67
Amygdala/hippocampus, total volume						
Right	6.14	0.62	5.87	0.85	6.56	0.67
Left	6.12	0.45	5.91	0.79	6.63	0.78
Total cranial volume	1,229	153	1,188	153	1,234	118

^aIn cubic centimeters.^bFor the prefrontal region measurements, N=22; for the caudate measurements, N=21.^cFor the prefrontal region and total cranial measurements, N=29.

deficit patient/normal subject comparison ($F=1.80$, $df=1, 43$, $p=0.19$). The differences in right and left caudate volumes for the deficit patients ($F=14.26$, $df=1, 16$, $p<0.002$) and the normal comparison subjects ($F=86.11$, $df=1, 29$, $p<0.001$) were highly significant. The addition of household socioeconomic status and total cranial volume as covariates did not significantly alter the pattern of results from the primary analyses.

DISCUSSION

The results of this study revealed a significant difference between the deficit and nondeficit schizophrenic patients in total right and left prefrontal volumes. The comparison of the deficit and nondeficit patients' brain region volumes with those of normal subjects revealed that these differences between the two schizophrenic subgroups were due to smaller prefrontal volumes in the nondeficit patients rather than greater volumes in the deficit patients. The differences in total prefrontal volumes between the two schizophrenic subgroups and between the nondeficit patients and the normal subjects were primarily due to smaller white matter volumes rather than gray matter volumes in the nondeficit patients. The finding of altered total prefrontal volume in the nondeficit rather than the deficit patients contradicts the study hypothesis. The two schizophrenic subgroups did not differ in caudate volumes, although the difference in right caudate volume approached a trend level ($F=2.68$, $df=1, 34$, $p=0.11$). The left caudate was significantly larger in the two schizophrenic subgroups than in the normal subjects. There was a trend for the right caudate to be larger in the deficit patients than in the normal subjects. There was no difference between

the nondeficit patients and the normal subjects in right caudate volume. Deficit patients and normal subjects, but not nondeficit patients, exhibited a significant right/left asymmetry in caudate volumes. Amygdala/hippocampus complex volumes did not differ between the two schizophrenic subgroups. As was the case with the total schizophrenic group (33), both schizophrenic subgroups differed from the normal comparison subjects in amygdala/hippocampus complex volume.

The failure to find a relation between prefrontal volume and deficit symptoms is consistent with the reports of Andreasen and associates (7, 20), who failed to find a relation between negative symptoms and the frontal lobe area. Pfefferbaum et al. (14) found a trend for negative symptoms to be inversely correlated with frontal sulcal size; i.e., patients with smaller sulci had more negative symptoms. In contrast, Ota et al. (12) observed a trend for negative symptoms to be associated with frontal lobe atrophy. No previous reports in the literature have examined the relation of caudate or amygdala/hippocampus complex volumes to negative symptoms.

Several methodological issues must be considered in evaluating the validity of the prefrontal and caudate findings. First, the region-of-interest analyses were corrected for total cranial volume, indicating that observed group differences were not due to differences in head size. The use of total cranial volume does not answer the question of whether the observed morphological changes are due to a generalized or a focal process. However, the pattern of results is not consistent with the proposition that the observed group differences are due to generalized tissue loss. Second, the separation of the total prefrontal volume measurements into gray and white matter measurements was performed on MRI images uncorrected for nonhomogeneous radio frequen-

cies. Nonhomogeneous radio frequencies could potentially affect the measurement of gray and white matter volumes, but there is no reason to believe that the MRIs of one of the schizophrenic subgroups or of the normal comparison subjects in this study were preferentially affected by such a lack of homogeneity. Third, the prefrontal cortex is a structurally and functionally heterogeneous region (48). The prefrontal region volume measured included both the orbital and the dorsolateral prefrontal cortex. The failure to measure these cortical areas and their subcomponents independently may have obscured the existence of differences between the schizophrenic subgroups in the volume of prefrontal region gray matter. The demarcation of specific prefrontal regions or gyri would enhance the ability to assess the possibility of differences in gray matter between the two groups. Fourth, caudate volumes were determined from coronal not transaxial images, and the lateral margins of the caudate can be difficult to distinguish on coronal images ($<1, 45^\circ$). The difficulty in discerning the lateral margins could potentially affect the accuracy of the caudate volume measurements, although acceptable reliability was achieved for this measurement. The use of transaxial images in future studies would alleviate this problem.

What are the implications of these results for the study hypothesis? The failure to find altered prefrontal volumes in deficit patients suggests that if structural changes in the prefrontal cortex are associated with the production of deficit symptoms, then either these structural changes occur in subcomponents of the prefrontal cortex or, alternatively, gross structural changes in the prefrontal cortex, detectable by MRI, are not associated with deficit psychopathology. The difference in prefrontal volume between the deficit and nondéficit patients appeared to be due largely to differences in white rather than gray matter. These results suggest the possibility that nondéficit patients, but not deficit patients, are characterized by fewer connections between the prefrontal cortex and other brain regions. If the observation of smaller white matter volume in nondéficit patients is confirmed, important questions arise concerning the nature of the pathophysiological process underlying the loss of white matter in nondéficit patients.

If structural changes in the dorsolateral prefrontal circuit are associated with the production of deficit symptoms, then the results of this study suggest that the caudate may be primarily involved. The caudate was bilaterally enlarged in the deficit patients. Deficit patients maintained the right/left asymmetry observed in normal subjects, whereas right/left asymmetry was lost in the nondéficit patients. These results suggest either that there may be a bilateral failure in the normal age-related reductions in the caudate in deficit patients (35) or that the caudate is enlarged secondary to the bilateral loss of neurons in some other brain region (49). The loss of right/left asymmetry and the enlargement of the left but not the right caudate in the nondéficit patients suggests that a process different from that occurring in

deficit patients underlies the changes in caudate volume in nondéficit patients.

This study represents our initial effort to test our structural hypothesis. Future studies examining the other components of the dorsolateral prefrontal circuit proposed by Alexander et al. (31) are required to determine the full extent to which structural changes in the circuit are or are not responsible for the production of deficit symptoms.

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Psychiatric Illnesses in Families of Subjects With Schizophrenia-Spectrum Personality Disorders: High Morbidity Risks for Unspecified Functional Psychoses and Schizophrenia

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Objective: The authors determined morbidity risks for psychiatric illnesses in the families of probands with schizophrenia-spectrum personality disorders. **Method:** Subjects were recruited from the community through newspaper advertisements. Subjects were identified as having schizophrenia-spectrum personality disorders ($N=30$) if they met at least three, four, or three DSM-III-R criteria for schizoid ($N=14$), schizotypal ($N=20$), and/or paranoid ($N=15$) personality disorder, respectively. The comparison subjects had no psychiatric diagnoses ($N=8$) or had other personality disorders ($N=12$); none of the subjects in either group had any DSM-III-R axis I diagnosis. Trained interviewers collected family history information about the relatives of the two groups; the interviewers were blind to the probands' diagnoses. **Results:** The risks for schizophrenia, other functional psychoses, and schizophrenia-spectrum personality disorders were significantly higher in the relatives of subjects with schizophrenia-spectrum personality disorders than in the families of the comparison subjects. **Conclusions:** The high rate of schizophrenia in the families of probands with schizophrenia-spectrum personality disorders is consistent with the previous findings of higher than normal rates of these personality disorders in the biological relatives of schizophrenic patients. The significance of the high rate of unspecified functional psychoses is unclear. Use of the family study method, by which valid differential diagnosis of psychoses is possible, is indicated. The results from the current study do not rule out the possibility that the schizophrenia-spectrum personality disorders are related to psychoses in general rather than specifically to schizophrenia.

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The concept of schizophrenia-spectrum personality disorders is derived from converging lines of evidence that suggest a familial link between certain personality styles and schizophrenia. The early investigators, who worked closely with the relatives of schizophrenic patients (1-3), provided extensive phenomenologic descriptions of these personality styles. Studies by Kety et al. (4) provided a basis for operational descriptions of these disorders, and, consequently, criteria for one of the schizophrenia-spectrum

diagnoses (i.e., schizotypal personality disorder) in DSM-III emerged (5). Later studies (6-9) by a number of investigators showed higher risks of schizophrenia-spectrum personality disorders, including schizotypal, paranoid, and schizoid personality disorders, in the families of schizophrenic patients than in the families of normal subjects, thus validating the concept of schizophrenia-spectrum personality disorders. The finding that the risk of schizophrenia is higher in the siblings of schizophrenic patients if the parents have schizotypal personality disorder (10) further supports the familial relationship between schizophrenia-spectrum personality disorders and schizophrenia. Finally, several neurobiological studies (11-13) have shown deficits in eye tracking, attention, and information processing in subjects with schizophrenia-spectrum personality disorders similar to those observed in schizophrenic patients.

If schizophrenia-spectrum personality disorders represent partial or complete penetrance of the schizophrenic gene(s), then a higher risk of schizophrenia in

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TABLE 1. Studies of Psychiatric Illnesses in Relatives of Subjects With Schizophrenia-Spectrum Personality Disorders

Study	Study Design	Findings in Relatives
Soloff and Millward (14)	Open study using family history method with probands with depression, schizophrenia, or schizotypal and borderline traits	Rates of schizophrenia not significantly different between groups
Torgersen (20)	Direct blind interviews of monozygotic co-twins of schizotypal or borderline patients	No cases of schizophrenia
Baron et al. (16)	Open study using family history method with relatives of schizotypal or borderline patient and comparison subjects	No cases of schizophrenia in relatives of schizotypal patients
Schulz et al. (17)	Open study using direct interviews of relatives of patients with schizotypal and borderline personality or depression	High rates of depression but not schizophrenia in relatives of patients with schizotypal and borderline personality
Schulz et al. (18)	Open study using family history method with relatives of patients with schizophrenia and patients with borderline personality and schizotypal traits	Rates of schizophrenia similar in the two groups
Lenzenweger and Loranger (21)	Blind study using family history method with schizotypal subjects ^a	Significantly higher rate of schizophrenia in relatives of schizotypal subjects than in comparison group
Siever et al. (19)	Blind study using family history method with patients with schizotypal and/or paranoid personality disorder	Higher risk for schizotypal personality but not schizophrenia in relatives of schizotypal or paranoid probands than in comparison group
Battaglia et al. (22)	Open study using family history method with schizotypal subjects	Higher risk for schizophrenia in relatives of schizotypal probands than in comparison group

^aSchizotypal subjects identified on the basis of Chapman's Scale of Perceptual Aberration.

the families of subjects with schizophrenia-spectrum personality disorders than in normal subjects can be expected. To date, few controlled studies have evaluated the risk of schizophrenia in such families (14–22). The data from these studies are inconsistent (see table 1) and have not unequivocally shown a significantly higher risk for schizophrenia in the families of subjects with DSM-III schizophrenia-spectrum personality disorders than in normal or comparison families. There are several possible reasons for this inconsistency. First, many studies used family history methods to assess the morbidity risks, and such methods are likely to have low sensitivity to psychiatric illnesses in family members. Second, considering the low prevalence of schizophrenia, the differences in morbidity risks between the schizophrenia-spectrum and normal families would be small and therefore difficult to detect. Third, schizophrenia-spectrum disorders are likely to be heterogeneous in their etiology, and only some of the cases would therefore be related to schizophrenia.

In the current study we attempted to overcome some of these methodological difficulties by recruiting a larger group of subjects from the community. Ideally one would prefer to directly interview all family members. However, since this is not practical with a large number of subjects, the family history method was used. In an effort to increase the sensitivity of the family history instrument, we added extensive probe questions to the family history form.

METHOD

Probands

Subjects with schizophrenia-spectrum personality disorders, subjects with other personality disorders,

and normal subjects were recruited from the community through newspaper advertisements. The newspaper advertisements recruited either normal subjects or individuals who had any one of the following: 1) "experience with ESP, clairvoyance, telepathy, 6th sense"; 2) "out of body experiences"; 3) "enjoy solitary activities or consider themselves 'loners'"; or 4) "consider close friendships to be unnecessary and overrated." The last two items (i.e., social isolation and lack of social desire) were included in the advertisement to recruit individuals with negative symptoms. Interviewers screened the initial inquiries on the telephone to exclude individuals with obvious major psychiatric illnesses or histories of drug abuse. Eligible subjects were then invited for direct interviews. Informed consent was obtained from all participants after the purpose of the study and the procedures involved were explained. Trained clinicians administered 1) the Schedule for Affective Disorders and Schizophrenia—Lifetime Version (SADS-L) (23) or the Structured Clinical Interview for DSM-III-R—Non-Patient Version (SCID-NP) (24) and 2) the Structured Interview for DSM-III Personality Disorders (original or revised version) (25). On the basis of the direct interviews and all other available information, best estimates of DSM-III-R axis I and axis II diagnoses were reached for all the participants in a diagnostic meeting. Individuals with axis I diagnoses were excluded from the study.

To recruit individuals with the broad spectrum of personality traits related to schizophrenia, individuals with schizoid and paranoid traits were included. It is possible that individuals who have moderately severe schizophrenia-related personality psychopathology and experience three or four of the traits in each diagnostic category may still not meet any of the diagnoses. In order to not lose these subjects or, more important, not include them in the comparison group, the thresholds

were lowered by one criterion. Thirty people met these criteria for schizoid (N=14), schizotypal (N=20), and/or paranoid (N=15) personality disorders. All but five met the full criteria required by DSM-III-R. Another 20 subjects had diagnoses of personality disorders other than schizophrenia-spectrum disorders (N=12) or did not have any diagnosis (N=8). The mean age of the subjects with schizophrenia-spectrum personality disorders was 31.6 years (SD=8.3), and the mean age of the comparison group was 33.6 years (SD=10.6). The two groups also had similar male-female ratios (17:13 and 10:10, respectively) and Hollingshead (26) socioeconomic levels (mean=2.89, SD=0.97, and mean=3.41, SD=1.06, respectively).

Family History Information

Interviewers obtained family history information by using the Family History Research Diagnostic Criteria (FH-RDC) (27). The FH-RDC format was modified by adding extensive probe questions for all items; both parts I and II of the modified FH-RDC were administered to all family members. Part I of the FH-RDC elicits demographic information and basic information concerning psychiatric treatment and hospitalization. Part II of the FH-RDC is used to determine specific axis I diagnoses and antisocial, borderline, and schizophrenia-spectrum personality disorders. Questions were added to the instrument to probe for each psychiatric symptom, such as hallucinations or deterioration in function. The purposes of adding specific questions to the interview were to standardize the probing and to increase the sensitivity of the instrument. Preliminary data from our laboratory (28) suggest that these modifications increased the sensitivity of the FH-RDC in detecting schizophrenia (copies of the modified FH-RDC are available on request). To obtain information regarding schizophrenia-related personality disorders in the family members, additional personality-related criteria were included in the FH-RDC. Again, extensive probe questions were added for each personality disorder criterion. A pilot study was conducted in which family history information was obtained by blind rates from one or two first-degree relatives of each of 20 subjects with schizophrenia-spectrum personality disorders and 30 individuals without such disorders. The majority of these subjects were obtained from another independent study group. We then compared the family-history-derived diagnoses with the diagnoses reached by direct interview with the Structured Interview for DSM-III Personality Disorders. The modified FH-RDC correctly identified 15 of the 20 subjects with schizophrenia-spectrum personality disorders (i.e., a sensitivity of 75%) and 26 of the 30 nonspectrum subjects (a specificity of 87%). The interrater reliabilities (kappas) for FH-RDC diagnoses of schizophrenia, unspecified functional psychoses, affective disorders, and antisocial personality disorder ranged from 0.65 to 0.89.

Trained interviewers (master's-level clinicians and a psychiatrist), who were blind to the probands' diagnoses,

collected family history information on both first- and second-degree relatives. The information was collected from the probands themselves. Because the subjects were volunteers and not patients, it was difficult to obtain information from second informants. However, in a minority of cases (33% of the schizophrenia-spectrum subjects and 25% of the comparison group), information was available from second informants. The information from the second informant allowed an increase in the ascertainment of about 18%. In addition, it clarified diagnostic questions in a few cases. The information on each family member was reviewed in a best-estimate diagnostic meeting chaired by a senior psychiatrist (G.T.), and blind FH-RDC diagnoses were given to each individual. Information was available for 321 (83.2%) of all adult (>15 years of age) first- and second-degree relatives of the subjects with schizophrenia-spectrum personality disorders; 129 relatives were first degree. Similarly, information was available for 248 (83.7%) of all first- and second-degree relatives of the comparison subjects; 101 were first degree.

Age-adjusted morbidity risks for psychiatric illnesses were calculated for both groups on the basis of Weinberg's short method (29). For the age correction, the risk periods were estimated to be 15 to 45 years for schizophrenia and related disorders, 15 to 70 years for depression, and 15 to 60 years for manic disorder. The chi-square test with Yates's correction and Fisher's exact test, when appropriate, were used to compare the risks of psychiatric illnesses in the two groups. We hypothesized that the morbidity risk for schizophrenia would be higher in the families of the subjects with schizophrenia-spectrum personality disorders than in the families of the comparison subjects, and therefore we carried out a one-tailed test to accept or reject the hypothesis. Statistical power determination indicated that the analyses had adequate power to detect differences given the number of subjects, an alpha level of 0.05 (one-tailed), and expected effect size. The effect size was estimated on the basis of the 5.4% rate of schizophrenia observed in first- and second-degree relatives (N=269) of schizophrenic patients in a pilot study using the same family history method.

RESULTS

Table 2 presents the rates of psychiatric diagnoses in the two groups, and table 3 gives the age-adjusted morbidity risks. Histories of schizophrenia were found in three first-degree relatives of subjects with schizophrenia-spectrum personality disorders and in none of the relatives of the comparison subjects. However, because of the low number of first-degree relatives, this difference was not statistically significant. When both first- and second-degree relatives were considered, the risk for schizophrenia was found to be significantly higher in the families of the schizophrenia-spectrum subjects than in the comparison families. In the larger cohort, FH-RDC diagnoses of schizophrenia were given to five

TABLE 2. Age-Corrected Risks of Psychiatric Illnesses in Relatives of Subjects With Schizophrenia-Spectrum Personality Disorders and Subjects With No Diagnoses or With Other Personality Disorders

FH-RDC Diagnosis	Relatives of Spectrum Probands ^a				Relatives of Comparison Probands ^a			
	First-Degree		First- and Second Degree		First-Degree		First- and Second-Degree	
	N	%	N	%	N	%	N	%
Schizophrenia	3	3.3	5	1.9 ^b	0	0.0	0	0.0
Unspecified functional psychoses	5	5.6	11	4.2 ^c	1	1.4	2	0.9
Schizophrenia-spectrum personality disorders	14	15.6	29	11.2 ^d	7	9.7	11	4.9
Depression	13	19.1	15	7.9	6	10.9	10	6.5
Bipolar disorder	2	2.7	4	1.9	1	1.6	1	0.6
Alcoholism	7	5.4	18	5.6	8	7.9	14	5.7
Anxiety disorder	3	2.3	3	0.9	1	1.0	1	0.4
Antisocial personality disorder	2	1.6	4	1.2	4	3.9	4	1.6

^aSee table 3 for age-corrected total numbers of relatives at risk.

^bSignificantly higher than rate for relatives of comparison probands ($p=0.03$, Fisher's exact test, one-tailed).

^cSignificantly higher than rate for relatives of comparison probands ($p=0.007$, Fisher's exact test, one-tailed).

^dSignificantly higher than rate for relatives of comparison probands ($\chi^2=5.51$, $df=1$, $p=0.009$, one-tailed).

of the 259 age-corrected at-risk relatives of the schizophrenia-spectrum subjects and none of the 225 age-corrected at-risk members of the comparison family group. All five cases of schizophrenia originated from different and unrelated families. Three cases of schizophrenia came from the families of subjects who met the criteria for schizoid personality disorder (in most cases they also had additional schizotypal and/or paranoid traits; estimated morbidity risk of 2.7%). Two cases (1.3%) were in relatives of schizophrenia-spectrum subjects without schizoid personality disorder.

Unspecified functional psychoses were also more frequent in the families of the schizophrenia-spectrum subjects than in the comparison families (table 2). Five first-degree relatives and six second-degree relatives of the schizophrenia-spectrum subjects had histories of functional psychoses. Only one first- and one second-degree relative of the comparison subjects experienced unspecified functional psychoses. The rates of unspecified functional psychoses in the families of spectrum subjects with and without diagnoses of schizoid personality were 3.7% and 4.7%, respectively.

Schizophrenia-spectrum personality disorders were also more prevalent in the first- and second-degree relatives of subjects with spectrum personality disorders than in the relatives of the comparison subjects (table 2). Other psychiatric illnesses, such as affective illness, antisocial personality disorder, and alcoholism, were equally prevalent in the two groups. None of the findings changed when the five schizophrenia-spectrum subjects who did not meet the full criteria were excluded.

DISCUSSION

These results indicate high risks of schizophrenia, other functional psychoses, and schizophrenia-spectrum personality disorders in relatives of research volunteers with diagnoses of schizophrenia-spectrum personality disorders. These risks were significantly higher

TABLE 3. Age-Corrected Number of Family Members at Risk of Various Psychiatric Illnesses in Families of Subjects With Schizophrenia-Spectrum Personality Disorders and Subjects With No Diagnoses or With Other Personality Disorders

FH-RDC Diagnosis	Number at Risk			
	Relatives of Spectrum Probands		Relatives of Comparison Probands	
	First-Degree	First- and Second-Degree	First-Degree	First- and Second-Degree
Schizophrenia and related disorders	90	259	72	225
Depression	68	189	55	155
Bipolar disorder	75	216	64	174
Other disorders	129	321	101	247

than those observed in the relatives of normal subjects and volunteers with other personality disorders. The risks for other major psychiatric illnesses, such as affective illness and substance abuse, were similar in the two groups.

One of the most striking findings of the study was the higher risk for unspecified functional psychoses in the relatives of the schizophrenia-spectrum subjects. It is likely that the lack of complete information made the diagnosis of schizophrenia difficult and that some family members who were diagnosed as having unspecified functional psychosis did, in fact, experience schizophrenic psychosis. Indeed, the family history method is inadequate for addressing this issue, and a family study method, by which a valid differential diagnosis of psychosis is possible through direct interviews of the relatives, is indicated. However, one can not rule out the possibility that these individuals experienced psychoses other than schizophrenia. A number of family members had each had one or two episodes of psychosis that lasted less than 6 months and were not followed by social, occupational, or other functional impairment. If

psychosis in general is more likely to occur in the families of subjects with schizophrenia-spectrum personality disorders than in the families of normal subjects, then the familial link between the schizophrenia-spectrum personality disorders and psychosis may be more pervasive and not limited to schizophrenia. In this context, we note that most studies have compared the prevalences of schizophrenia-spectrum personality disorders in families of schizophrenic probands and families of normal subjects; only a few studies have compared the families of schizophrenic subjects with families of patients with other psychotic illnesses. Studies that included relatives of patients with other psychotic illnesses showed higher rates of schizotypal personality disorder in these groups than in relatives of normal subjects (30, 31).

Schizophrenia was also more frequent in the relatives of the subjects with schizophrenia-spectrum personality disorders than in the relatives of the comparison group. This difference was significant only when both first- and second-degree relatives were considered. This finding is consistent with the report by Schulz et al. (23), who found similar risks for schizophrenia in families of patients with borderline personality disorder and schizotypal traits and in families of schizophrenic patients. Lenzenweger and Loranger (21) also found a higher rate of schizophrenia in the families of schizotypal patients than in comparison subjects. Lenzenweger and Loranger identified schizotypal patients on the basis of high scores on Chapman's Scale of Perceptual Aberration. Siever et al. (19) reported a higher rate of schizophrenia in the first-degree relatives of patients with schizotypal and/or paranoid personality disorder than in their comparison group, although the difference was not statistically significant. Another recent open study (22) also showed a significantly higher risk of schizophrenia in families of schizotypal individuals than in comparison families.

It should be noted that the group of individuals with schizophrenia-related personality traits recruited in the current study may not be representative of the population of such individuals. The findings reported here may not be generalizable and may be an artifact of the sampling method. For instance, one can argue that subjects with schizophrenia-spectrum personality disorders who had schizophrenic relatives were likely to respond to the advertisement out of curiosity about their own mild, but schizophrenia-like, experiences. However, one can make a similar argument for clinical samples, since schizotypal individuals with schizophrenic relatives are as likely to seek treatment. Alternatively, the unique clinical characteristics of the schizophrenia-spectrum subjects collected through a newspaper advertisement may explain the stronger finding in our study than in the previous studies. In contrast to the previous investigations, the current study actively recruited individuals with negative symptoms, such as low social desire or social isolation. Consequently, about one-half of the schizophrenia-spectrum subjects met three or more diagnostic criteria for schizoid personality disorder.

Thus, relatively more negative symptoms in the schizophrenia-spectrum subjects in the current study than in the previous studies may be responsible for the higher rate of schizophrenia in their relatives. Schizophrenia-spectrum personality disorders are essentially descriptive syndromes derived from a classification system that is based on phenomenology rather than pathophysiology or etiology. Therefore, these disorders are likely to be heterogeneous in etiology, and only a subgroup of them may be related to schizophrenia. The risk of schizophrenia in the relatives of subjects with schizophrenia-spectrum personality disorders may therefore vary and depends on the characteristics of the study group. A number of investigators (20, 29, 32, 33) have argued that negative symptoms, such as social isolation and restricted affect, form the core phenomenology that is related to the genetic factors in schizophrenia. Subjects with schizophrenia-spectrum personality disorders and negative symptoms are therefore likely to have a familial etiology and to have families with high rates of schizophrenia.

Our finding of a significantly higher risk of schizophrenia in the families of subjects with schizophrenia-spectrum personality disorders than in the families of subjects who do not have psychiatric diagnoses or have other personality disorders supports the hypothesis that there is a familial link between schizophrenia and schizophrenia-spectrum personality disorders. The risk for unspecified functional psychoses, which may or may not be related to schizophrenia, was also higher in the families of the schizophrenia-spectrum subjects than in the families of the comparison subjects. The implications of this finding are not clear, but the possibility that the schizophrenia-spectrum personality disorders are related to psychoses in general, rather than specifically to schizophrenia, cannot be ruled out.

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Length of Stay and Recidivism in Schizophrenia: A Study of Public Psychiatric Hospital Patients

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Objective: Psychiatric beds in public hospitals have decreased 80% since 1955, but admissions have risen correspondingly, largely because of high recidivism rates. Decreases in numbers of beds have been partly achieved by shortening the length of stay, which lessened by half between 1970 and 1980. This study was undertaken to determine whether duration of hospital treatment affects the rate and rapidity of relapse among schizophrenic patients. **Method:** Data on 1,500 patients from 10 state hospitals were gathered for 18 months after initial discharge. Predictor variables included age, sex, marital status, race, number of previous admissions, location of the facility, and length of stay. Data were analyzed by survival analysis with a Cox regression model for two times to initial relapse: 30 days and 18 months (outcome). **Results:** Length of stay was significantly related to each time to relapse after the effects of number of previous admissions and age were partialled out. Facility location was not predictive, but intrahospital effects were tested by examining the data on the largest facility; again, length of stay significantly predicted relapse. **Conclusions:** Although the magnitude of the effect was small, the clinical significance of the findings is the greater likelihood that brief-stay patients will be rehospitalized within 30 days after discharge than will patients treated for longer periods. Brief hospitalization seems generally applicable to psychiatric populations, but there may be a small but important group of seriously mentally ill patients for whom other alternatives are possibly more appropriate and should be explored.

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In spite of growing criticism (1-4), mental hospitals have continued to decrease their patient populations, though at a much slower pace than in the past decade (5, 6). Since 1955 psychiatric beds in public hospitals have decreased 80%, but the number of admissions, due largely to high rates of recidivism (1), has risen almost 90% (unpublished tables, National Institute of Mental Health, 1989). The reduction in beds has been achieved in part by shortening the length of stay. Nationwide, the median public hospital stay dropped

by almost half between 1970 and 1980 (7)—to 28 days in 1986 (8). In Illinois, where this study was carried out, the median stay dropped from 35 days in 1970 to 14 days by 1986 (information from the Office of Information Services, Department of Mental Health and Developmental Disabilities, Springfield, Ill., 1989).

Although such a brief stay may be consistent with a philosophy of deinstitutionalization, the evidence to support this practice is not generalizable to all patient groups (9). Attempts to identify patients who might benefit clinically from longer inpatient care (10) have not been very fruitful (11). Critics, however, often overlook the fact that disaffiliated or resistive patients either are ineligible or generally refuse to participate in after-care or alternative programs (12).

Separate lines of research have addressed the effects of length of stay on readmission rates. Controlled investigations have generally concluded that the two are unrelated (13-28), whereas epidemiological and large-scale studies have tended to find that shorter hospital stays are associated with high return rates (29-36). This discrepancy may be essentially methodologic and attributable to differences in sample selection, the defini-

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tion of brief treatment, and criteria for readmission. Neither group of studies truly examined the complexity of the variables involved (37), having attended mainly to the frequency of relapse.

Because of the importance of this issue and the absence of studies that assess how other variables affect the risk of readmission at any point in time, we undertook a retrospective study to examine the relation between hospital stay and subsequent length of community survival of patients admitted to a public mental health system. The purpose of this study was to determine whether length of hospital stay affects the rate and rapidity of relapse for seriously mentally ill individuals. Specifically, it was designed to test the hypothesis that for schizophrenic patients discharged from state hospitals, time in the community before relapse is directly related to duration of hospital treatment.

METHOD

Fifteen hundred schizophrenic patients were selected at random from the 10 state hospitals in Illinois that serve the acutely mentally ill (a research institute was excluded). Some facilities served the city of Chicago, and others were located in small rural communities; some were small (30–50 acute care beds with about 275 annual admissions) and others large (200–300 beds and 4,000–6,000 admissions). The annual turnover ranged from seven to 21 patients per acute care bed, and the median length of stay ranged from 12 to 75 days. Fifty subjects were chosen from each of the two smallest hospitals, and the other 1,400 subjects represented 26.4% of 5,300 patients discharged from the remaining eight institutions. According to state policy, all patients receiving direct discharges are referred upon release to state-aided community aftercare programs.

The final database contained 3,281 discharge records of the 1,500 patients, including those for the index release and for 18 months afterward. Predictor variables other than length of stay (covariates) in the records included age at discharge, sex, marital status (married versus other), race (white versus other), number of previous episodes, and location of discharging facility (urban versus nonurban). Time to relapse (i.e., number of days from index discharge to initial rehospitalization) was the outcome studied.

Since traditional multiple linear regression models are not applicable to time-to-event data of this type, a survival analysis with the Cox proportional hazard regression model (38) was used to describe the relation between the covariates and time to relapse; only covariates with a significance of $p \leq 0.10$ were included. Separate analyses were conducted for the entire 18-month period and for the first 30 days after discharge to examine more clearly the indications of rapid readmission.

It should be noted that the hospital and length of stay variables are confounded. Ideally, length of stay would be randomly assigned to patients within hospitals, and hospitals would be matched on median length of stay.

To adjust for hospital (as either a fixed or a random effect) would undesirably remove much of the variability in length of stay. Alternatively, the overall analysis could show a significant association due only to differences in hospital policy. For this reason, we analyzed data for the entire study group as well as for subjects from the largest hospital ($N=369$), where a sufficient number of patients were available to replicate the entire analysis. If length of stay is significantly related to readmission rates in both analyses, we can more confidently conclude that it is a reliable predictor of readmission both within and between hospitals.

In this study, time in the community started with release and ended with relapse; a readmission was the binary outcome variable of interest and a continuous community stay for the entire study period was a censored event. Releases or relapses within the same day were considered to be equal to a half-day (coded 0.5). Log transformation was used for the number of previous admissions to minimize the impact of outliers. Two separate regression analyses were done to examine length of stay: a log transformation of the actual number of days and a five-category classification of the original values. BMDP software programs (39) were used to perform the analyses.

RESULTS

The mean age of the 1,500 study subjects was 35.4 years ($SD=11.7$). They were predominantly unmarried men (68% were male) and were roughly equally divided between white and nonwhite. The mean number of previous admissions to a state facility was 5.4 ($SD=7.0$); first admissions constituted 23% of the sample. During the 18 months, the subjects had an average of 1.6 hospitalizations. The median length of stay for the index hospitalization was 17 days; 90% of the subjects were released within 3 months, and just 3% stayed more than 6 months. Over 53% of the subjects relapsed during the study period: 14% within a month, 34% within 6 months, and 47% within a year. The average time in the community before the initial relapse was 340 days (median=447).

Table 1 presents data on relapse rates for five categories of length of stay at the two major follow-up points (30 days and 18 months) as well as at 6 months and 1 year. The data suggest a linear relation between length of stay and rate of relapse; that is, at each time point, subjects with shorter hospitalizations tended to have higher rates of relapse than those with longer stays. Thus, the relapse rate 30 days after discharge was 16.5% for patients with stays of 7 days or fewer, compared to about 10% for those who stayed between 1 and 2 months. At 6 months the rates were 39% and 30%, respectively, and at 1 year or more the return rates were roughly 50% regardless of length of stay.

Table 2 summarizes the stepwise Cox regression analysis for the five-category classification of length of stay. Findings, though, were almost identical when

TABLE 1. Relapse Rates of State Hospital Patients According to Length of Stay During Index Hospitalization

Length of Hospital Stay ^a	N	Patients Who Relapsed Within 30 Days ^b		Patients Who Relapsed Within 6 Months ^b		Patients Who Relapsed Within 1 Year ^b		Patients Who Relapsed Within 18 Months ^b	
		N	%	N	%	N	%	N	%
≤7 days	316	52	16.5	123	38.9	150	47.5	170	53.8
8–14 days	352	56	15.9	124	35.2	168	47.7	194	55.1
15–30 days	343	53	15.0	122	35.6	163	47.5	187	54.5
31–60 days	232	23	9.9	70	30.2	105	45.3	121	52.2
> 60 days	257	29	10.9	70	27.2	112	43.4	127	49.4
Total	1,500	213	14.2	509	33.9	698	46.5	799	53.3

^aStays of 1 week or less and 8–14 days are considered brief stays; a stay of 15–30 days, about average; 31–60 days, longer than average; and beyond 60 days, an extended stay.

^bTime to relapse was measured from day of discharge to day of rehospitalization.

TABLE 2. Results of Cox Regression Analysis for Time to Relapse of State Hospital Patients^a

Stepwise Model Covariate	Relapse Within 30 Days			Relapse Within 18 Months		
	Coefficient	SE	z	Coefficient	SE	z
Number of previous hospitalizations	0.531	0.070	7.39 ^b	0.565	0.038	14.88 ^b
Age	-0.028	0.007	-3.86 ^b	-0.022	0.003	-6.36 ^b
Sex	0.306	0.144	2.12 ^c	—	—	—
Length of stay	-0.400	0.161	-2.50 ^d	-0.190	0.083	-2.34 ^c

^aTime to relapse was measured from day of discharge to day of rehospitalization.

^bp<0.001.

^cp<0.05.

^dp<0.01.

length of stay was used as a continuous variable ($z=2.29$, $p=0.02$, for overall survival at 18 months with length of stay added to the equation).

In the analyses, number of previous hospitalizations stood out as the most significant factor influencing time to relapse. The next most significant factor was patients' age. After accounting for the significant effects of these two variables—which were always chosen first by the stepwise procedure—length of stay emerged as a significant predictor of relapse in the regression model. Thus, a shorter-hospital stay significantly contributed to higher readmission rates at 1 and 18 months. (Results were similar for analyses of stays of up to 6 and 12 months.) Figure 1 shows the time to relapse curves for three lengths of stay: a week or less, 2 weeks to a month, and more than 60 days.

Marital status, race, sex, and location of the facility (urban versus nonurban) were the remaining covariates. Marital status was not significant at any of the relapse times. Sex proved to be significant for stays up to 30 days but not up to 18 months: men returned to the hospital more rapidly than women within the first 30 days. Finally, both race and location of the facility were significant at 18 months, but not after length of stay was added to the equation.

The largest facility was selected for the last analyses (using the five length of stay categories) to determine whether the findings of the regression model reflected not just interhospital but also intrahospital effects. The results were the same as in the previous analysis. After the effects of previous admissions and age were par-

tialed out, length of stay was again significantly related to a time to relapse of up to 30 days ($z=2.21$, $p<0.03$) and 18 months ($z=1.97$, $p<0.05$).

DISCUSSION

Before we discuss the significance of our results, certain methodologic considerations require attention. This study was limited to schizophrenic patients in public psychiatric settings, and any generalization to other disorders or settings may not be warranted. The data are retrospective, preventing causal connections, and the scope of the study was confined to the variables available in the data registry. Also, because of the absence of clinical ratings and information about substance abuse, we do not know whether length of stay had anything to do with levels of psychopathology at discharge or during subsequent readmissions or was confounded by problems of dual diagnosis.

Our major finding is that in a cohort of 1,500 schizophrenic patients tracked retrospectively over an 18-month period, length of hospital stay was significantly related to length of time in the community between discharge and readmission. This result is similar to that reported in earlier large-scale studies (29, 32, 33, 40), which used samples with multiple diagnoses and often drew conclusions based on global trends or rates.

Perhaps the most clinically significant finding is that schizophrenic patients hospitalized for short stays were more likely to return within 30 days after discharge

than patients who had been treated for longer periods. Even in controlled studies (14, 19), readmissions within a month were greater among patients who had brief treatment. Such rapid relapse after discharge is more likely related to the nature of the previous hospitalization than is relapse months or years after discharge.

Although the relation between length of stay and relapse is statistically significant, the magnitude of the effect is small. For stays of 14 days or fewer, the readmission rate was 16.2%, and for stays over 30 days, it was 10.6% (a 5.6% difference). At 6 months the return rates were 37.0% and 28.6%, respectively (an 8.4% difference), and at 1 year and 18 months the differences were about 3.5%. But again, most clinically important is the reduction in the rate of early relapsers, who are probably responsible, in large part, for many readmissions and excess resource utilization.

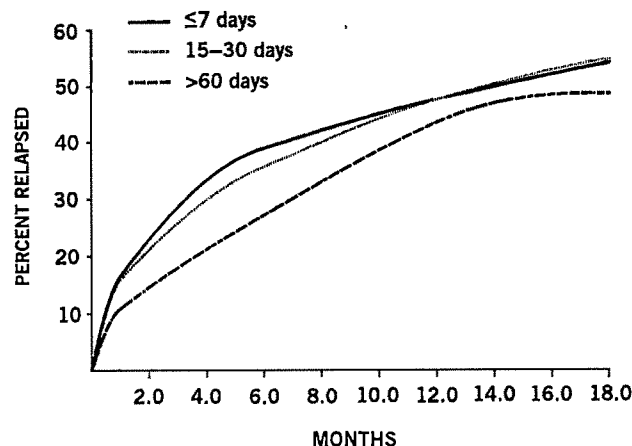
Previous hospitalizations, sex, and age also independently predicted early readmission. Our data extend the evidence for the well-documented relation between psychiatric history and recidivism (41) by showing that number of previous hospitalizations is also inversely associated to time in the community between relapses. The finding that men return to the hospital more rapidly than women (at least during the first 30 days after discharge) is consistent with other research (33, 40). Considerable data about repeat users of hospital facilities (12, 42, 43) further point to a preponderance of male patients, perhaps because of their nomadic qualities and disengaged behavior.

Research comparing relapse rates of younger and older patients is inconsistent (29, 36, 42, 44). Also, many studies were conducted when geriatric patients constituted a large proportion of psychiatric admissions (29, 40). Only 2% of our study group was over 65 years of age. And even though lack of social support is generally linked to recidivism (45), it is not surprising that marital status failed to predict relapse, since 87% of the 1,500 subjects were single.

Finally, Caton (46) found that interhospital difference (based on length of stay and amount of neuroleptic medication administered) was a significant predictor of relapse; other comparative investigations (32, 34) have reported substantially higher readmission rates at facilities where patients are more rapidly discharged. In our study the three largest institutions, with the briefest length of stay (median=12 days), served urban dwellers, while the smaller hospitals, with lower turnover (median length of stay=35 days), served smaller cities and suburban or rural areas, seriously confounding these variables. When length of stay was controlled, however, rural/urban differences did not predict early relapse. Furthermore, the relation between length of stay and time in the community was not just an artifact of interhospital variability, since we were able to demonstrate a similar relationship within a single facility.

The overall finding of this study raises questions concerning the role of brief hospitalization in psychiatric treatment. The issue is not the general applicability of brief treatment but whether there may be a small but important group of patients for whom a brief stay is

FIGURE 1. Time to Relapse of Patients With Varying Lengths of Stay at State Hospitals^a



^aTime to relapse (measured from day of discharge to day of rehospitalization) is plotted at 0.1-month intervals. Of the 1,500 patients, 316 (21%) stayed in the hospital a week or less, 343 (23%) stayed 15-30 days, and 257 (17%) stayed more than 60 days.

associated with short community tenure and for whom the benefit of lengthier hospitalization (10, 47) or a residential alternative (48) is an option that needs to be systematically explored both at the policy level and through further research.

Policy considerations should address the human cost inherent in the situation of patients who are unresponsive to briefer therapies. Research into this subject needs to be geared toward understanding the variables that might mediate a relation between length of stay and relapse, as well as those aspects of the public mental health system that actually determine length of stay (49). The drop in median length of stay in Illinois, for example, undoubtedly reflects the wide imbalance between beds and admissions (50), which is increased by the fact that fewer private resources are available. However, even if it is more economical, repeated hospitalization is extremely disruptive to continuity of care and quality of life and is potentially demoralizing to caretakers.

In theory, controlled studies are best able to address these issues, but in practice, random assignment to study groups is not suitable for severely ill individuals (51). Naturalistic research is a likely substitute if it can be designed prospectively to make reliable assessments of diagnosis, psychopathology, use of psychotropic drugs, level of social support, and quality of the discharge environment (52). Such efforts to develop clinical profiles of problematic patients should result in earlier identification of high-risk individuals and treatment/intervention aimed at prolonging community stay (53).

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Relative Performance of For-Profit Psychiatric Hospitals in Investor-Owned Systems and Nonprofit Psychiatric Hospitals

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Objective: The authors analyzed the differences in operational and financial performance between 42 matched pairs of for-profit psychiatric hospitals belonging to multifacility organizations and nonprofit psychiatric hospitals for the fiscal years ending in 1986 through 1990. **Method:** The pairs of short-term hospitals were matched according to location, standard metropolitan statistical area, or wage index. Analyses were based on data on these hospitals from the Health Care Financing Administration. The groups of variables studied included the hospitals' operational performance and productivity, profitability and payer mix, revenue and expenses, and capital structure. Differences in the mean values of the variables for the for-profit hospitals and the nonprofit hospitals were analyzed by pairwise *t* tests. **Results:** The for-profit organization hospitals had significantly higher net revenue, lower salary expenses, and higher profits than the nonprofit hospitals. Patients in the for-profit hospitals had longer stays, and these hospitals had fewer full-time employees per adjusted inpatient day and per adjusted discharge. **Conclusions:** The higher prices and operating margins of the for-profit hospitals belonging to investor-owned systems reflect the profit-maximizing goal of these facilities. The ability of for-profit organization hospitals to achieve economies of scale in expenses, however, was not evident except in the case of salary expenses.

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Dorwart and Schlesinger (1) noted an increasing privatization of inpatient mental health services. According to the American Hospital Association, private nonprofit and for-profit hospitals constituted 45% of the 534 inpatient psychiatric facilities in 1980 (2). In 1988 over 64% of the 726 inpatient facilities were privately owned (3). Much of the growth in privately owned facilities has been in hospitals owned by multifacility for-profit organizations. From 1987 to 1989 the National Association of Private Psychiatric Hospitals (4) reported a 28% increase in the number of hospitals in multifacility for-profit corporations (from 147 to 188). The number of independent for-profit facilities decreased by approximately 7% (from 42 to 39), and the number of not-for-profit hospitals increased by only 5% (from 64 to 67) during this time period.

As with acute care hospitals, observers have expressed concerns that the financial goals of the for-profit hospitals owned by multifacility organizations

will affect access to care and the cost of care (1, 5). Since the financial goal of these hospitals is to maximize the wealth of shareholders, they have incentives to price their services aggressively, select profitable patients, and keep expenses low. The purpose of this study was to determine whether short-term nonprofit psychiatric hospitals and for-profit psychiatric hospital systems differ in revenue, price, expenses, and other performance measures. A study of this nature is especially timely given the recent charges that the profit motive has adversely influenced the behavior of some large for-profit psychiatric hospitals belonging to investor-owned systems (6, 7).

METHOD

Data

The data for the short-term psychiatric hospitals studied were derived primarily from the Health Care Financing Administration's Minimum Cost Data tapes for the fiscal periods ending in 1986 through 1990. In contrast to the data available for acute care hospitals, case-mix indexes for psychiatric specialty hospitals are not available from the Health Care Financing Administration. However, to decrease the heterogeneity of the case mix, the study was limited to short-term inpatient psychiatric

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hospitals. A short-term hospital is defined by the American Hospital Association as a hospital in which the average length of stay is under 30 days or in which more than 50% of all patients are admitted to units where the average length of stay is under 30 days (3).

To control for market variables that might influence the performance of inpatient psychiatric hospitals, we matched nonprofit hospitals with hospitals belonging to for-profit systems by using three criteria. We first attempted to match on the basis of location in the same county. If we were unable to match a hospital by using this criterion, we then matched according to whether the hospital was located in a standard metropolitan statistical area or not. Hospitals that were not matched according to one of these two criteria were matched to hospitals on the basis of similar wage indexes. Data were obtained for the matching process from 1) the American Hospital Association's 1989 annual survey of hospitals (8), 2) the 1987 and 1991 editions of the *Directory of Investor-Owned Hospitals* (9, 10), and 3) the 1989 *Area Resource File* (Office of Data Analysis and Management, Bureau of Health Professions, Washington, D.C.).

We were able to match seven hospitals in the same county. Thirty-three hospitals were matched using the standard metropolitan statistical area criterion, and two hospitals were matched on the basis of wage index. The matching process was validated by testing the means of pair differences for a set of market measures. We found no significant differences between the nonprofit and for-profit pairs in wage index, county population, number of psychiatric beds in the county, and the following age population categories: population per thousand less than 15 years of age, population per thousand between 15 and 24 years of age, and population per thousand between 25 and 34 years of age. In addition, there were no significant differences in number of beds.

Complete data were available for only 42 (63%) of the 67 short-term nonprofit psychiatric hospitals reported by the National Association of Private Psychiatric Hospitals in 1989 (4). Since data were available for only seven nonprofit multifacility system hospitals, an analysis of this system-owned group was not feasible.

The sample of 42 for-profit system-owned hospitals represents 22% of the 188 for-profit hospitals owned by multihospital systems reported by the National Association of Private Psychiatric Hospitals in 1989 (4). It should be noted, however, that the National Association of Private Psychiatric Hospitals does not report the number of short-term hospitals owned by systems, and not all private psychiatric hospitals are members of the National Association of Private Psychiatric Hospitals.

Dependent Variables

The dependent variables used in the study are commonly examined measures of operating and financial performance. We separated these variables into four types: operational performance and productivity, profitability and payer mix, revenue and expenses, and capital structure.

Operational performance measures include occupancy rate and length of stay. The productivity measures are discharges per bed, full-time employees per daily census, and total asset turnover. The last variable measures the efficiency of a firm in using assets to produce revenue. We would expect profit-maximizing for-profit hospitals to have a higher occupancy rate, a longer length of stay, greater utilization of assets, and a lower number of full-time employees.

Commonly used variables that show a firm's overall profitability in accounting and cash terms were used to measure each hospital's profitability. To assess differences in the selection of patients (patient mix), we separately examined the proportions of the hospitals' patients who were Medicaid and Medicare beneficiaries. We would expect higher profitability and a higher proportion of more generous (nongovernment) payers among for-profit hospitals than among nonprofit hospitals. For-profit providers of care have an incentive to increase revenue and minimize expenses. However, even though purchases from affiliated organizations may increase a hospital's expenses in order to increase the affiliate's revenue, such purchases are a relatively small part of a hospital's budget, and the overall corporate goal is to maximize profits, so we did not expect these exceptions to affect our results.

To examine further any differences in profitability, we evaluated total net patient revenue (total patient revenue less deductions for contractual allowances, charity care, and bad debt) and total patient expenses, as well as components of total expenses. Net patient revenue reflects both pricing and discounts granted. These measures, along with cash flow per bed (discussed below), were recast in 1986 dollars with the use of the Consumer Price Index.

All of the revenue and expense measures were examined on a per-discharge and a per-day basis. Neither net patient revenue nor expenses could be separated into inpatient and outpatient components. Therefore, we adjusted both the number of inpatient days and the number of inpatient discharges for outpatient utilization by multiplying by the ratio of gross revenue (both inpatient and outpatient) to gross inpatient revenue. Gross revenue is revenue before deductions for allowances, bad debt, and charity care.

To determine whether system affiliation of for-profit hospitals enabled them to achieve greater access to debt capital, the study used two ratio measures: long-term debt to net fixed assets and total debt to total assets. These measures reflect the amount of debt financing used. Since long-term debt is typically used to finance long-lived assets, such as buildings and equipment, the ratio of long-term debt to fixed assets (net of accumulated depreciation) indicates the proportion of these assets financed with debt. Similarly, the ratio of total debt to total assets indicates the proportion of total assets financed with short- and long-term debt.

The study also measured the ability to pay debt. Capital

TABLE 1. Operational Performance and Productivity of Matched Pairs of Nonprofit and For-Profit Psychiatric Hospitals in Fiscal Years 1986–1990^a

Variable	Number of Hospital Pairs	Nonprofit Hospitals		For-Profit Hospitals		Mean Difference Between Hospital Pairs	Analysis	
		Mean	SD	Mean	SD		t (df=41)	p
Number of beds	42	78.58	56.60	90.19	35.68	-11.61	1.36	n.s.
Occupancy rate ^b	42	0.70	0.17	0.66	0.17	0.04	1.31	n.s.
Length of stay (days) ^c	42	18.44	6.57	23.71	4.47	-5.27	4.35	<0.01
Discharges per bed ^d	42	15.11	6.89	10.53	3.19	4.58	3.77	<0.01
Full-time employees per daily census ^e	42	3.65	1.45	2.53	0.94	1.12	3.86	<0.01
Total asset turnover ratio ^f	42	2.72	8.99	3.79	7.13	-1.07	0.59	n.s.

^aBased on data from the Health Care Financing Administration's Minimum Cost Data tapes.^bAverage daily census divided by available beds.^cNumber of inpatient days divided by number of discharges.^dNumber of discharges divided by number of beds.^eAverage number of full-time employees divided by adjusted inpatient days/365. Adjusted inpatient days=inpatient days × (inpatient revenue + outpatient revenue)/inpatient revenue.^fNet patient revenue plus other operating revenue divided by total assets.

tal and depreciation expenses as a proportion of total expenses relate capital costs to a firm's budget. Capital expenses are defined as Medicare allowable expenses and include interest, leasing, and depreciation expenses. Finally, the ratio of cash flow to total debt relates cash available to the firm's total debt.

Sixty-two percent (N=26) of the 42 individual nonprofit hospitals had reported data for a 4-year period, while 14% (N=6) had reported data for a 3-year period, 12% (N=5) for a 2-year period, and 12% for only 1 year. Seventy-four percent (N=31) of the 42 for-profit system-owned hospitals had reported data for a 4-year period, 17% (N=7) for a 3-year period, and 9% (N=4) for a 2-year period. For hospitals with data on more than 1 year, we computed averages to eliminate the effects of extreme values.

For the statistical analysis we used pairwise t tests to compare differences between the mean values of the variables for the for-profit organization-owned hospitals and the nonprofit hospitals. The procedure tests whether the differences are significantly different from zero.

RESULTS

Tables 1–4 present the results of the tests of the pairwise differences for the operational and financial performance variables.

Operational Performance and Productivity

Table 1 shows that there were significant differences between the nonprofit and for-profit hospitals in length of stay, number of discharges per bed, and number of full-time employees per adjusted census. During the study period, the average stay was 5 days longer in the for-profit hospitals than in the nonprofit hospitals. Occupancy rates, however, did not differ significantly. For-profit hospitals averaged four fewer discharges per

bed, and as expected, they used about one less full-time employee per adjusted patient day than did the nonprofit hospitals.

Surprisingly, the total asset turnover ratio was not higher for the for-profit hospitals. At first glance, it appears that the for-profit hospitals were not more successful in using total assets to produce revenue. However, as will be reported later, the assets in the for-profit hospitals are newer. When their value is recorded at historical cost, as is required, the newer assets would tend to lower the total asset turnover ratio. Thus, the absence of significant differences in this ratio may be partially explained by differences in asset ages.

Profitability and Payer Mix

Table 2 shows the results of the pairwise tests of differences in the profitability and payer mix variables. As expected, the two types of inpatient psychiatric hospitals differed significantly during the study period in total profit margin, return on assets, and percentage of Medicare patients. The nonprofit hospitals averaged a 46% lower total profit margin and a 14% lower return on total assets than the for-profit system-owned group.

The nonprofit hospitals served a slightly higher percentage of Medicare patients during the study period as well. However, the proportion of Medicaid patients did not differ significantly between the two types of hospitals.

Revenue and Expenses

Table 3 shows that there were significant differences between the matched pairs in net revenue per adjusted day and per adjusted discharge, in outpatient revenue as a proportion of total net patient revenue, in salary expenses per adjusted day and per adjusted discharge, and in total expenses per adjusted discharge.

The nonprofit hospitals generated about \$86 less in net patient revenue per day and \$3,060 less in net pa-

TABLE 2. Profitability and Payer Mix of Matched Pairs of Nonprofit and For-Profit Psychiatric Hospitals in Fiscal Years 1986-1990^a

Variable	Number of Hospital Pairs	Nonprofit Hospitals		For-Profit Hospitals		Mean Difference Between Hospital Pairs	Analysis		
		Mean	SD	Mean	SD		t	df	p
Total profit margin ^b	42	-0.55	0.83	-0.09	0.46	-0.46	4.00	41	<0.01
Return on assets ^c	42	-0.09	0.41	0.05	0.31	-0.14	2.15	41	<0.05
Cash flow per bed (dollars) ^d	42	2,349.59	11,351.07	6,755.91	11,014.46	-4,126.32	1.67	41	n.s.
Percentage of Medicaid patients ^e	39	0.07	0.13	0.04	0.08	0.03	1.30	38	n.s.
Percentage of Medicare patients ^f	39	0.20	0.08	0.16	0.08	0.04	2.83	38	<0.01

^aBased on data from the Health Care Financing Administration's Minimum Cost Data tapes.^bNet income divided by net patient revenue.^cNet income divided by total assets.^dNet income plus depreciation expenses divided by number of beds.^eMedicaid discharges divided by total discharges.^fMedicare discharges divided by total discharges.TABLE 3. Revenue and Expenses of Matched Pairs of Nonprofit and For-Profit Psychiatric Hospitals in Fiscal Years 1986-1990^a

Variable	Number of Hospital Pairs	Nonprofit Hospitals		For-Profit Hospitals		Mean Difference Between Hospital Pairs	Analysis	
		Mean	SD	Mean	SD		t (df=41)	p
Net patient revenue per adjusted inpatient day (dollars) ^b	42	252.20	96.15	338.47	58.87	-86.27	4.80	<0.01
Net patient revenue per adjusted discharge (dollars) ^c	42	4,913.26	2,924.43	7,373.46	1,928.33	-3,060.20	4.80	<0.01
Outpatient revenue as percentage of total patient revenue	42	0.40	0.47	0.01	0.03	0.38	5.18	<0.01
Total expenses per adjusted inpatient day (dollars) ^b	42	329.63	131.56	348.48	84.56	-18.84	0.44	n.s.
Total expenses per adjusted discharge (dollars) ^c	42	6,216.05	3,748.80	8,107.99	2,223.17	-1,891.94	2.87	<0.01
Routine expenses per adjusted inpatient day (dollars) ^b	42	82.28	47.84	122.28	45.86	-40.00	0.43	n.s.
Routine expenses per adjusted discharge (dollars) ^c	42	1,575.93	1,350.73	1,924.09	526.89	-348.16	1.49	n.s.
Salary expenses per adjusted inpatient day (dollars) ^b	42	215.11	84.71	218.97	42.32	-3.86	5.97	<0.01
Salary expenses per adjusted discharge (dollars) ^c	42	3,942.07	2,016.28	4,759.00	959.64	-816.93	3.33	<0.01
Ancillary expenses per adjusted inpatient day (dollars) ^b	42	25.16	41.02	24.14	12.69	1.02	0.87	n.s.
Ancillary expenses per adjusted discharge (dollars) ^c	42	514.28	1,073.17	539.29	272.43	-25.01	0.15	n.s.

^aBased on data from the Health Care Financing Administration's Minimum Cost Data tapes.^bAdjusted inpatient days=inpatient days × (inpatient revenue + outpatient revenue)/inpatient revenue.^cAdjusted discharges=discharges × (inpatient revenue + outpatient revenue)/inpatient revenue.

tient revenue per adjusted discharge than the for-profit system-owned hospitals, which is consistent with shorter length of stay. In addition, they derived a greater proportion of their patient revenue from outpatient services than did the for-profit hospitals.

To maximize profits, one would expect for-profit hospitals to minimize costs. However, the nonprofit hospitals incurred higher expenses than the for-profit hospitals only in the case of salaries. They incurred \$56 more in salary expenses per adjusted day and \$1,183 in salary expenses per adjusted discharge than the for-profit hospitals. As shown in table 1, the nonprofit hospitals used more full-time employees per patient day.

On a per-discharge basis, total expenses were higher

in for-profit facilities, which is probably an outgrowth of longer length of stay. Evidently, for-profit system-owned hospitals did not create any economies of scale with respect to total costs. The lack of significant differences in routine expenses per day and per discharge suggests that nonprofit psychiatric hospitals saved on expenses other than salaries.

Capital Structure

The findings presented in table 4 show that the two types of hospitals differed significantly with respect to the amount of debt financing they used and their ability to pay debt. As indicated by the ratio of total debt to

TABLE 4. Capital Structure of Matched Pairs of Nonprofit and For-Profit Psychiatric Hospitals in Fiscal Years 1986–1990^a

Variable	Number of Hospital Pairs	Nonprofit Hospitals		For-Profit Hospitals		Mean Difference Between Hospital Pairs	Analysis		
		Mean	SD	Mean	SD		t	df	p
Ratio of long-term debt to net fixed assets	39	0.71	0.82	1.04	1.90	-0.33	0.69	38	n.s.
Ratio of total debt to total assets	41	0.52	0.44	0.81	0.60	-0.29	1.98	40	<0.05
Ratio of capital expenses to total expenses ^b	42	0.06	0.04	0.10	0.06	-0.04	3.24	41	<0.01
Ratio of depreciation expenses to total expenses	42	0.04	0.02	0.07	0.05	-0.03	3.02	41	<0.01
Ratio of cash flow to total debt ^c	41	0.03	0.72	0.26	0.90	-0.23	1.72	40	<0.10

^aBased on data from the Health Care Financing Administration's Minimum Cost Data tapes.

^bRatio of total Medicare allowable capital expenses (interest, leasing, depreciation) to total operating expenses.

^cRatio of net income plus depreciation expenses to total debt.

total assets, the for-profit system-owned hospitals had a higher amount of debt than the nonprofit hospitals. The key factor behind an organization's borrowing capability is its ability to generate the funds, specifically cash, to pay the debt. Therefore, the high debt position of the for-profit hospitals is probably supported by these hospitals' ability to generate the cash flow to meet debt service, as reflected by the higher ratio of cash flow to total debt, which is marginally significant.

Consistent with these findings, the nonprofit hospitals had significantly lower ratios of capital expenses and depreciation expenses to total operating expenses. Since annual depreciation expenses are generally lower for older facilities, these lower ratios may indicate that the nonprofit hospitals had older facilities than the for-profit hospitals. It is also possible that their leasing expenses or interest rates were lower as well. Interest expenses would be lower especially if the nonprofit hospitals had tax-exempt debt.

CONCLUSIONS

In today's health care environment, cost control for mental health services is becoming a major goal of employers and insurers. In our study of matched pairs of for-profit hospitals in multifacility systems and nonprofit hospitals, we found that the for-profit hospitals had higher total net patient revenue per day and per discharge than similar nonprofit hospitals. The higher total net patient revenue per adjusted inpatient day may reflect higher prices or differences in payer mix. More specifically, the for-profit hospitals served fewer Medicare patients than the nonprofit hospitals but approximately the same proportion of Medicaid patients. The lower proportion of Medicare patients may reflect decisions to accept fewer nongenerous government payers and more profitable private payers.

In addition to higher net revenue per day, the for-profit hospitals were characterized by higher net revenue per discharge. The latter is the result of prices and perhaps of payer mix or a longer average length of stay. Higher revenue from longer stays may imply that for-profit system-owned hospitals are prolonging the fewer patient stays at their facilities to increase revenue. How-

ever, despite the significantly greater length of stay at for-profit hospitals, we found no significant difference between for-profit and nonprofit hospitals with respect to occupancy rate.

Higher revenue per day and per discharge would reflect the primary financial goal of for-profit organizations—to maximize the wealth of shareholders. Indeed, the results of this study show that the for-profit system-owned hospitals achieved significantly higher profitability than the nonprofit hospitals during the late 1980s. Not only did the for-profit hospitals realize higher revenue, they also had lower salary expenses per patient day, which appears to have been achieved through reduced staffing levels. The greater number of hospitals owned by the for-profit systems may have enabled them to develop economies in staffing or select less complex cases, which in turn resulted in lower costs. An alternative explanation is that the longer stay is associated with a lower severity of illness during the last part of the stay. Lower staffing needs during this part of the stay would work to decrease the average staffing level.

However, the for-profit system-owned hospitals did not differ significantly from the nonprofit hospitals in expenses for routine care and ancillary services. In fact, the for-profit hospitals had a higher total cost of care per discharge than the nonprofit hospitals. Thus, the profit-maximization goal was not achieved through cost minimization by the for-profit system-owned hospitals. We note, however, that cost-based reimbursement, which is still prevalent for inpatient psychiatric hospitals, provides incentives to increase, not lower, costs (11).

The for-profit hospitals did differ from the nonprofit hospitals in their capital structure. Since most of them are members of large for-profit organizations, system size and reputation provided greater access to debt capital. The higher depreciation expenses as a proportion of total expenses among the for-profit hospitals indicates that their facilities possessed newer equipment or buildings or that they had renovated their facilities more recently than the nonprofit hospitals.

In order to compete with for-profit hospitals, nonprofit hospitals may need to renovate and refurbish their facilities. However, their poor profitability and higher salary costs may restrict their ability to use both

internal capital (profits) and external capital to update their facilities. Internal funds may not be available, and external capital suppliers are likely to view these facilities as poor investments. Thus, nonprofit inpatient psychiatric hospitals may be at risk of closing or changing behavior to emulate the more financially successful for-profit systems.

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Enhanced Suppression of Cortisol Following Dexamethasone Administration in Posttraumatic Stress Disorder

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Objective: The authors investigated the possibility of enhanced negative feedback sensitivity of the hypothalamic-pituitary-adrenal (HPA) axis in posttraumatic stress disorder (PTSD) by using a low dose of dexamethasone. **Method:** Baseline blood samples were obtained at 8:00 a.m., and 0.5 mg of dexamethasone was administered to 21 male patients with PTSD and 12 normal age-comparable men at 11:00 p.m. Cortisol and dexamethasone levels were measured 9 and 17 hours after dexamethasone administration. **Results:** After correction for differences in dexamethasone levels, the PTSD patients showed greater suppression of cortisol in response to dexamethasone than did the normal subjects. This was true even in patients meeting concurrent diagnostic criteria for major depression. **Conclusions:** The data support earlier studies showing that HPA abnormalities in PTSD are different from those seen in depression and suggest that the low-dose dexamethasone suppression test may be a potentially useful tool for differentiating the two syndromes and further exploring differences in their pathophysiology. (Am J Psychiatry 1993; 150:83–86)

There is considerable debate concerning the nature of hypothalamic-pituitary-adrenal (HPA) abnormalities in posttraumatic stress disorder (PTSD). Our work has provided evidence for lower than normal HPA activity, possibly secondary to enhanced negative glucocorticoid feedback sensitivity. We have reported both lower mean 24-hour urinary cortisol excretion (1, 2) and more lymphocyte glucocorticoid receptors (3) in patients with PTSD than in normal subjects. These findings are opposite of the hypercortisolemia reported in major depression.

In contrast to the aforementioned findings, others have suggested that the HPA axis may be overactivated in PTSD in a manner similar to that observed in depression. One study showed greater than normal urinary cortisol excretion (4), and another demonstrated blunted ACTH response to corticotropin-releasing factor (5). Data from dexamethasone suppression test (DST) studies of PTSD patients have been inconclusive in regard to abnormal cortisol metabolism in PTSD. Of

the four published reports (6–9), all indicated normal suppression of cortisol in nondepressed PTSD patients given the standard DST. In PTSD patients meeting concurrent criteria for major depression, two studies (6, 7) showed a nonsuppressive response to dexamethasone in some patients, and two studies (8, 9) showed normal suppression in this subgroup.

To further explore HPA axis dysfunction in PTSD patients, we administered a low dose of dexamethasone to patients and normal subjects. Given the preliminary data from our laboratory suggesting the possibility of more effective feedback inhibition, we hypothesized that rather than showing the classic nonsuppression observed in many patients with major depression, PTSD patients would suppress cortisol to a greater extent than normal subjects in response to dexamethasone. If so, the standard 1-mg dose, which almost completely suppresses the normal cortisol response, would be too high to effectively discriminate normal from subnormal responses. In the present pilot study, 0.5 mg of dexamethasone, which produces more modest suppression in normal subjects, was used to explore the possibility of enhanced suppression of cortisol after dexamethasone administration in PTSD patients with and without comorbid major depression.

METHOD

The subjects were 21 nonmedicated male Vietnam combat veterans with PTSD (mean age=41.9 years, SD=2.4, range=38–48) and 12 normal age-comparable

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TABLE 1. Mean Cortisol and Dexamethasone Blood Levels After 11:00 p.m. Administration of 0.5 mg of Dexamethasone in Male Vietnam Veterans With PTSD and Age-Comparable Normal Men

Group	Baseline		Cortisol ($\mu\text{g/dl}$) ^a				Dexamethasone (ng/ml) ^b			
			8:00 a.m.		4:00 p.m.		8:00 a.m.		4:00 p.m.	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Normal (N=12)	15.09	4.17	4.78	2.93	4.51	2.28	1.46	0.48	0.53	0.28
PTSD (N=21)	14.29	3.70	1.78 ^c	1.45	1.89 ^c	1.36	2.19 ^c	0.64	0.86 ^c	0.32
With major depression (N=11)	14.14	4.02	2.07 ^c	1.83	1.83 ^c	1.26	2.35 ^c	0.66	0.88 ^c	0.33
Nondepressed (N=10)	14.49	3.39	1.41 ^c	0.60	1.96 ^c	1.57	1.98	0.64	0.84	0.34

^aMeans of cortisol values at 8:00 a.m. and 4:00 p.m. were 1.45 and 1.25 $\mu\text{g/dl}$, respectively.

^bMeans of dexamethasone values at 8:00 a.m. and 4:00 p.m. were 0.41 and 0.22 ng/ml, respectively.

^cSignificantly different from value for normal subjects ($p < 0.05$, two-tailed, Tukey's honest significance difference test).

men (mean age=41.0 years, SD=6.2, range=30–49; all gave written informed consent. The patients were recruited from a specialized inpatient PTSD program at a U.S. Department of Veterans Affairs medical center. The normal subjects were largely hospital personnel, students, and acquaintances who responded to advertisements within the medical center. The normal subjects were screened for axis I disorders and for family history of psychiatric illness. All patients were free from psychoactive substance abuse, as confirmed by urine toxicology screens, for at least 1 month before testing. The patients were also free from major medical, endocrinological, psychotic, and organic illness as determined by history, physical examination, and routine clinical laboratory tests, including thyroid and liver function tests.

The patients were interviewed with the Structured Clinical Interview for DSM-III-R (SCID) (10). After diagnostic evaluation, the patients were further subdivided into those meeting and not meeting concurrent DSM-III-R criteria for major depression. Severity of PTSD was determined with the Mississippi Scale for Combat-Related Posttraumatic Stress Disorder (11), and depressive symptoms were assessed by using the 21-item Hamilton Rating Scale for Depression (12).

Baseline blood samples were obtained at 8:00 a.m. on the morning of the DST. At 11:00 p.m. each subject received an oral dose of 0.5 mg of dexamethasone. Blood samples for determination of cortisol and dexamethasone levels were obtained at 8:00 a.m. and 4:00 p.m. the following day. Plasma cortisol levels were determined with a commercially available radioimmunoassay kit, as previously described (2, 3). The inter- and intra-assay coefficients of variation for this method in our laboratory are 6.8% and 4.0%, respectively. Dexamethasone levels were measured by radioimmunoassay using a commercially available antibody (IgG Corporation, Nashville, Tenn.), as previously described (13). This assay can sensitively quantify dexamethasone levels of 0.20 ng/ml. The inter- and intra-assay coefficients of variation for this procedure are 8.2% and 6.0%.

Because of an a priori assumption that PTSD patients would show lower than normal baseline cortisol levels, the data were subjected to two-way repeated measures multivariate analysis of covariance (MANCOVA) (Group by Time) with baseline cortisol levels used as

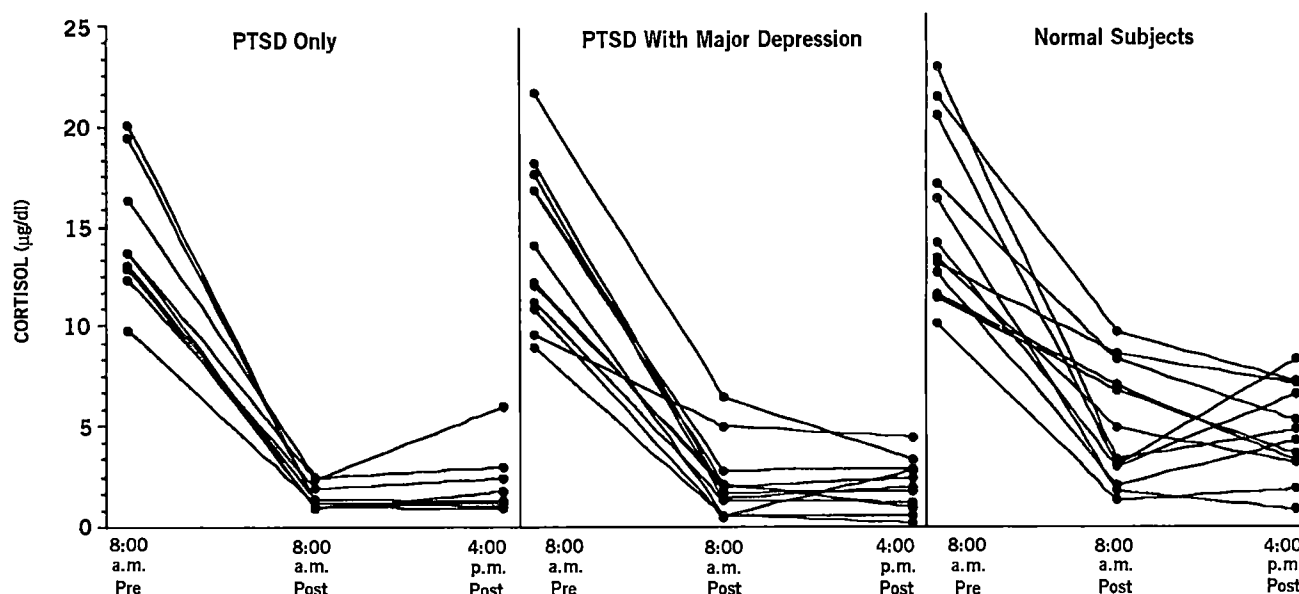
covariates. For this analysis all PTSD patients were combined into one group and compared to the normal subjects. The dexamethasone data were analyzed by using a two-way repeated measures MANOVA (Group by Time). Because of group differences in dexamethasone levels, the cortisol data were reanalyzed by using the dexamethasone values at 8:00 a.m. and 4:00 p.m. as covariates to correct for differences in dexamethasone levels (13). Next, the PTSD patients were subdivided into those with and without major depression, and they were compared to the normal subjects by means of the aforementioned analyses. Post hoc testing was performed by using the Tukey's honest significance difference test (two-tailed), at an alpha level of $p < 0.05$. Differences in clinical symptoms between the PTSD subgroups with and without major depression were evaluated by using Student's *t* test, two-tailed. Statistical analyses were performed on a microcomputer with SPSS (14).

RESULTS

Table 1 summarizes the biological data. In an initial analysis comparing the PTSD group as a whole to the normal subjects, MANCOVA revealed a significant group difference in the cortisol response to dexamethasone when the baseline cortisol values were used as covariates ($F=21.2$, $df=1, 30$, $p < 0.0001$). Dexamethasone levels were also found to be significantly different in the two groups ($F=11.37$, $df=1, 31$, $p < 0.002$). After correction of the cortisol data for differences in dexamethasone levels the PTSD patients still showed significantly greater cortisol suppression in response to 0.5 mg of dexamethasone ($F=9.01$, $df=1, 30$, $p < 0.005$).

A secondary analysis was performed in which the PTSD patients were subdivided on the basis of the presence or absence of comorbid major depression. MANCOVA revealed an overall main effect of group in the cortisol response to dexamethasone ($F=10.39$, $df=2, 29$, $p < 0.001$), reflecting the fact that the cortisol response to dexamethasone in both PTSD subgroups differed from that of the normal subjects. However, post hoc testing revealed no significant differences in cortisol between the PTSD subgroups (table 1). In contrast, post hoc testing revealed that the significant main effect for

FIGURE 1. Individual Cortisol Blood Levels After 11:00 p.m. Administration of 0.5 mg of Dexamethasone in Male Vietnam Veterans With PTSD and Age-Comparable Normal Men



group ($F=6.26$, $df=2, 30$, $p<0.005$) in dexamethasone levels reflected a significantly higher drug level only in the PTSD patients with concurrent major depression. The dexamethasone levels of the PTSD patients without depression were comparable to those of the comparison subjects. When the cortisol data were again analyzed by using dexamethasone levels as covariates to control for differences in dexamethasone pharmacokinetics, MANCOVA revealed a significant main effect of group ($F=4.9$, $df=2, 29$, $p<0.01$). Post hoc testing revealed that the PTSD subgroups were comparable with respect to cortisol levels but that both groups' cortisol secretion was lower than that of the normal subjects (figure 1).

There were no significant correlations between liver enzymes and suppression of cortisol in the PTSD group (SGPT: $r=0.06$, $df=18$; alkaline phosphatase: $r=0.05$, $df=18$; LDH: $r=0.06$, $df=17$; SGOT: $r=0.09$, $df=18$).

There were no significant differences in severity of PTSD, as reflected by the Mississippi scale scores, between the PTSD patients with and without major depression (with depression: mean=134.7, $SD=16.0$; without depression: mean=130.0, $SD=10.5$). The Mississippi scale scores in both groups were in the severe range. There were also no significant differences in Hamilton depression scores between the two PTSD subgroups (with depression: mean=28.3, $SD=7.2$; without depression: mean=21.8, $SD=12.6$).

DISCUSSION

By using a low dose of dexamethasone it was possible to determine that rather than showing a normal or non-suppression response to dexamethasone, as reported by other investigators (6–9), PTSD patients show an exaggerated

suppression response to dexamethasone regardless of whether or not they concurrently meet diagnostic criteria for major depression. These results are consistent with our previous reports of low mean basal 24-hour urinary cortisol excretion (1, 2) and a higher than normal number of lymphocyte glucocorticoid receptors (3) in PTSD with and without major depression, and they support the developing hypothesis of an abnormally sensitive negative feedback system in the HPA axis (15).

Indeed, if the number of steroid receptors in the PTSD group were higher than in the normal subjects, as determined in the previous study, this would be consistent with the "supersuppression" in response to dexamethasone observed in the present study. A greater than normal availability of steroid receptors would theoretically allow for enhanced migration of the steroid receptor complex into the cell nucleus, where the steroid could exert genomic effects, resulting in a compensatory decrease in cortisol production. Basal and postdexamethasone glucocorticoid receptors are currently being measured in our laboratory to directly address this question.

An exaggerated cortisol response to dexamethasone could also occur in cases where dexamethasone bioavailability is greater than normal. In the present study PTSD patients showed a higher mean dexamethasone level than the normal subjects. However, the greater cortisol suppression in response to dexamethasone in the present study is not likely to have resulted solely from differences in dexamethasone levels because 1) correcting the cortisol data for differences in dexamethasone levels still resulted in significant group differences in postdexamethasone cortisol levels between the PTSD patients and normal subjects, and 2) dexamethasone levels were only significantly higher in the PTSD group with depression, whereas the two PTSD

subgroups showed comparable cortisol responses to dexamethasone and, in fact, the PTSD group without depression showed a slightly more dramatic response. Dose-response pharmacokinetic studies measuring dexamethasone levels at earlier time points after dexamethasone administration in a larger group of PTSD and normal subjects are clearly needed to further explore this issue.

Differences in dexamethasone bioavailability can result from a number of factors, including abnormalities in hepatic metabolism (13, 16) and medication effects (17). Specifically, in a study of patients with liver damage (16), the plasma half-life of dexamethasone was longer than normal. Given that most of the patients in the present study had histories of alcoholism, it is possible that long-term changes in hepatic function account for some of the present findings. Of note, Caron et al. (18) reported that recently detoxified alcoholics with abnormal liver function had lower postdexamethasone cortisol levels than did alcoholics with normal liver function. Although examination of the raw data revealed no obvious relationships between liver enzymes (SGOT, SGPT, LDH, and alkaline phosphatase) and cortisol suppression in the subjects in the present study, it is difficult to comment on the effect of liver function on DST results in the present study because the patients differed substantially on time since last drink, and liver function tests were performed anywhere from a week to a month before the DST. Medication effects on dexamethasone pharmacokinetics are unlikely, as all patients were medication free. Other factors that may account for differences in dexamethasone bioavailability, such as differences in the metabolism of dexamethasone by other tissue, such as lymphocyte and kidney (13), should be explored in subsequent studies. The present findings are consistent with reports of large interindividual differences in dexamethasone metabolism (13, 19) and support the use of indexes of dexamethasone bioavailability in studies of cortisol responsiveness to dexamethasone (19).

In the present study, the two PTSD subgroups were comparable with respect to severity of PTSD and severity of depression. That the PTSD patients with depression had Hamilton depression scores in the same range as those of the PTSD patients without depression underscores the observation that PTSD inpatients may be quite depressed regardless of whether they meet the full DSM-III-R criteria for major depression (20). Additionally, given the large symptom overlap between PTSD and depression (in symptoms of insomnia, impaired concentration, loss of interest, and social withdrawal), the high Hamilton scores in the PTSD patients may reflect severity of PTSD as well as depression. In any event, the lack of a significant difference in clinical symptoms between the two subgroups is consistent with the lack of difference in cortisol suppression after dexamethasone administration.

The present findings should be considered preliminary because of the small number of subjects; however, they may have important clinical implications. Many patients

with PTSD also meet diagnostic criteria for major depression. However, it is presently unclear whether the frequent co-occurrence of depression represents a true melancholia or a different dysthymic disorder that may be unique to PTSD. The current data support the latter hypothesis. Thus, the low-dose DST may be a potentially useful clinical and experimental tool for teasing apart these two syndromes and for further exploring their distinct pathophysiologic abnormalities.

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Anticipatory Stress in Children and Adolescents

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***Objective:** Circumstances surrounding the New Madrid earthquake prediction on Dec. 3, 1990, offered a unique opportunity to study the effects of a disaster warning stage on children and adolescents. **Method:** An initial structured interview was administered to 553 third- and 10th-grade students before December 3, with follow-up interviews conducted 6–8 weeks later. **Results:** This study documents the existence of a mild but prevalent PTSD-like reaction that arose from exposure to a prediction of disaster. **Conclusions:** Further study of anticipatory stress reactions is needed to provide insights into the development of methods for providing support to children during disaster warnings.*

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A major earthquake along the New Madrid fault line was predicted to occur on Dec. 3, 1990. (The fault runs from Marked Tree, Ark., to Cairo, Ill.) Although earthquake experts insist that no scientific method exists for reliably predicting earthquakes, the prediction received daily media coverage, relief agencies released information regarding earthquake preparedness, and several schools canceled classes for December 3. The effect of this prediction on the children who live along the New Madrid fault is unknown.

Disaster research provides insights into physical and mental health stressors that accompany widespread death and destruction. In addition to describing a typology of disasters, several authors conceptualize disasters as occurring in stages including warning and threat, impact, assessment and rescue, and recovery or long-term effects (1, 2). The warning stage of a disaster is characterized by denial or overreaction (2); little research has focused on this stage. Circumstances surrounding the earthquake prediction offered a unique opportunity to study the effects of the warning stage on children and adolescents.

The response of individuals to the various stages of a disaster covers a wide range of human emotions, including distress, anxiety, fear, and apprehension. A disaster represents an external stimulus or stressor, distress is a response to the stressor, and anxiety is the discomfort that accompanies the anticipation of or un-

certainty about an event (3). Fear and apprehension are realistic interpretations of the danger, and coping refers to individual differences in managing the stimulus and emotional response (4). The emotions experienced, as well as the coping strategies used, can be either adaptive or maladaptive.

Because of the earthquake prediction, the mid-South braced itself for a major disaster that never occurred. Previous research demonstrates, however, that psychiatric symptoms of stress occur not only because of the impact of a disaster, but also from mere exposure to trauma (5). Electronic media coverage of events such as disasters presents more provocative and potentially traumatic material. Vivid media images simulate actual traumatic conditions, and some individuals are predisposed to react symptomatically to the vulnerability portrayed. The main difference between this and actual trauma is that the viewer remains at a distance from harm's reach (6). This concept has precedence in law. "Zone of danger" cases allow persons to recover damages for psychic injuries that result from proximity to perilous situations (7).

Many conditions defining psychic trauma in children are also present in anticipation of disaster; they include threats against the child's life, fears about physical harm, concern over the safety of attachment figures, and misinformation or isolation surrounding different threats and fears (8, 9). Under these conditions, many victims develop symptoms of posttraumatic stress disorder (PTSD) including reexperiencing the traumatic event, avoidance of stimuli associated with the trauma, general numbing of responsiveness, and increased arousal (DSM-III-R). Terr (10–12) described four symptoms characteristic of all patients with PTSD: visualization, reenactment, trauma-specific fears, and sense of foreshortened future. In addition, specific symptoms such as posttraumatic play and contagion of

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posttraumatic symptoms (11) are seen. Clinical studies consistently list the following symptoms: development of trauma-related or mundane fears, sleep disturbances, nightmares, regressive bed-wetting, somatic complaints, acting out or withdrawal behavior, depressive behavior, mistrust, and irritability (9, 10, 13, 14).

Current theories view the severity of PTSD on a continuum, with level of trauma experienced (proximity to violence, degree of threat to life) and individual vulnerabilities as primary factors contributing to development of the disorder. Studies demonstrate that the number, severity, and duration of PTSD symptoms exhibited are primarily and directly related to the intensity of the traumatic experience (14–16).

Given less extreme stressors, a conceptual relationship exists between the reaction to stress and trauma and the individual's coping strategies and social support system (17–19). Coping strategies appear to play a mediating role in an individual's response to stress (20). According to the hardiness concept theory, adaptive mechanisms such as internal locus of control, high level of commitment to task, and anticipation of change as challenging combine to improve one's ability to deal with adversity.

The results of studies on stress reactions in children are varied and inconclusive. On the basis of studies from World War II, it was concluded that children do not experience anxiety in response to stressful events directly but display anxious symptoms in response to parental anxiety. This concept continues to be debated (3). Indeed, the study of stress, anxiety, and coping in children continues to be refined, and studies of children's responses to disaster test some of the basic concepts regarding stress and anxiety.

In order to explore the psychological effects of disaster prediction and planning on children and adolescents, a priori hypotheses included the following: 1) exposure to the prediction of an earthquake would result in symptoms commonly associated with PTSD, including increased anxiety, enactments or play related to the trauma, a decreased sense of personal safety, a sense of absence of a future, and ambivalence concerning where they live; and 2) involvement with preparations for disaster (an index of locus of control) and communication regarding concerns about the earthquake (an index of support) would be related to lower levels of anticipatory stress (17, 20, 21).

METHOD

In order to begin testing hypotheses, a group of latency-age children (third-graders) and adolescents (10th-graders) were interviewed before Dec. 3, 1990 (time 1), and 6–8 weeks later (time 2). These two groups were chosen to represent different developmental accomplishments and to be consistent with other trauma research (L. Terr, personal communication). The two groups were obtained in Memphis, Tenn., and Marked Tree, Ark., providing examples of an urban setting and a rural setting in the fault area.

Permission to conduct research was granted by the Memphis City and Marked Tree schools and several private schools; the county school system denied our request. Following this, a random number table was used to rank order the schools available. Each school principal was then contacted to obtain access to the third- and 10th-grade classrooms (14 elementary and 10 high schools). Informed consent by the students' parents was required for participation in the study. An average of 31% of students per classroom completed the interview.

Each student was individually interviewed by mental health workers who used a brief, highly structured interview designed for this study. The interview covered seven areas consistent with theories related to trauma and its effects: anxiety, personal safety, concerns about property, orientation to the future, disaster planning, religious orientation, and ability to plan for the future.

Each interview contained two sections; one focused on generalized anxiety and attitudinal questions, and the second focused on the earthquake prediction. Two initial interviews were used to control for effects of the instrument. The control interview included only the first section, which did not mention the earthquake. No control interview was administered at time 2. At both interviews, each student was asked to draw a picture of his or her city as imagined or experienced on Dec. 3, 1990.

Interviewers were university faculty, staff, and graduate students who had participated in a training session to review interview procedures. Student responses were recorded verbatim, and interviewers were instructed not to deviate from the interview format. All interviews were scored by one author (S.H.) after the establishment of reliability between two independent raters ($\kappa=0.96$) (S.H., L.K.).

The total study group size for the initial administration of the interview was 553 students (137 served as control subjects). Because of absences, transfers, and conflicting schedules, 494 follow-up interviews were conducted, representing an attrition rate of 10.7%. No significant differences were found on demographic or anxiety variables between those subjects lost to attrition and the remaining subjects, or between the control subjects and noncontrol subjects. Subsequent analyses used only noncontrol subjects who had complete responses at times 1 and 2 ($N=314$).

In addition, an anticipatory stress index was created by combining the following PTSD-like symptoms assessed during the interviews: earthquake- or trauma-related play, making drawings depicting earthquake scenes, sleep disturbances, nightmares related to the earthquake or other related trauma, fears, somatic complaints, recent nervous habits, preoccupation with the earthquake prediction, limited orientation to the future (negative or ambivalent responses to the idea of marrying or having children, limited or uncertain response regarding longevity), and concerns about personal and environmental safety. This index yielded a total possible score of 15.

RESULTS

Anticipatory Stress

In order to convey the impact of the prediction, the following statistics from the first interview are presented for review: of the 416 children and adolescents questioned about the prediction, 94.0% had heard about the December 3 prediction, 78.5% reported spending time thinking about an earthquake, over 50.0% reported planning for the disaster, and 35.9% stated that they believed something would happen on December 3.

Before December 3, the children and adolescents interviewed expressed concerns in the following ways. They had trouble sleeping at night and changed their bedtime routine. Some experienced insomnia, some rearranged their rooms in anticipation of an earthquake, and some reported earthquake-related sleep problems (i.e., awakening with fears about an earthquake happening, afraid to go to sleep because an earthquake might occur). Many experienced recent, repetitive dreams or nightmares and said that they had been afraid of something recently.

Results indicate an overall decrease in anxiety symptoms from times 1 to 2. At follow-up, significantly fewer children and adolescents reported sleep problems, nightmares, somatic concerns, and fears than before December 3 ($p < 0.05$). Table 1 summarizes the study group's responses at times 1 and 2.

Other symptoms commonly associated with PTSD were also affected by the prediction. Enactments were significantly less common after December 3, and earthquake-related play decreased significantly from time 1 to time 2 ($p < 0.05$).

Although many subjects expressed concerns about personal safety before December 3, no significant increase in feelings of personal safety was reported when the earthquake did not occur. However, results indicate subtle shifts in the children's and adolescents' attitudes toward the future. No significant differences were found between times 1 and 2 in the number of subjects who planned to marry and have children. However, in terms of life expectancy, a significantly greater number of subjects at follow-up felt that they would live into their seventies, and fewer subjects chose not to answer the question ($p < 0.05$).

Frequently, people who have experienced trauma develop a dislike or avoidance of the environment in which the trauma occurred. Thus, it was hypothesized that in anticipation of a major disaster, children and adolescents would be less positive about their home and community. Indeed, positive attitudes toward the mid-South rose significantly after December 3 ($p < 0.05$).

Finally, results of analyses that used the anticipatory stress index are presented. The most frequently endorsed items on this index included thinking about an earthquake and worries about the safety of home and school, as opposed to a shortened life expectancy and concerns about personal and parental safety, which

TABLE 1. Characteristics and Symptoms of 314 Children and Adolescents Interviewed Before (Time 1) and After (Time 2) the Predicted Date of the New Madrid Earthquake

Item	Time 1		Time 2		χ^2 (df=1) ^a
	N	%	N	%	
Games					
Earthquake-related	9	2.9	1	0.3	6.13 ^b
Trauma-related	13	4.1	7	2.2	1.25
Sleep					
Problems	120	38.2	78	24.8	18.57 ^b
Change in bedtime routine	83	26.4	93	29.6	1.55
Insomnia	49	15.7	28	8.9	7.55 ^b
Earthquake-related problems	19	6.1	1	0.3	16.06 ^b
Nightmares or bad dreams	101	32.2	72	22.9	10.74 ^b
Earthquake-related	24	7.6	3	1.0	19.05 ^b
Trauma-related	59	18.8	46	14.6	2.22
Movie-related	18	5.7	20	6.4	0.17
Fears/afraid of things	153	48.7	104	33.1	18.73 ^b
Weather	7	2.2	1	0.3	3.13
Own death	11	3.5	3	1.0	4.08 ^b
Death of other	10	3.2	7	2.2	0.24
Earthquake	64	20.4	8	2.5	47.27 ^b
Movie-related	13	4.1	12	3.8	0.00
War	0	0.0	25	8.0	19.36 ^b
Buildings falling	3	1.0	0	0.0	1.33
New habits	135	43.0	125	39.8	0.81
Somatic complaints	156	49.7	130	41.4	5.68 ^b
Belief that people can predict future	62	19.7	48	15.3	2.73
Plans to marry	231	73.6	239	76.1	1.45
Plans to have children	217	69.1	211	67.2	0.40
Life expectancy					
70 years or older	208	66.2	231	73.6	6.47 ^b
Does not know	34	10.8	16	5.1	8.50 ^b
Science helps make plans and predictions	240	76.4	246	78.3	0.70
News changes feelings	183	58.3	193	61.5	1.27
Positive feelings for mid-South	169	53.9	214	68.3	19.85 ^b
Sense of safety					
Personal	265	84.4	260	82.8	0.30
Parental	268	85.4	255	81.2	2.44
House	99	31.5	99	31.5	0.01
School	131	41.7	125	39.8	0.30
Belief in God	310	98.7	312	99.4	2.25
God controls things	270	86.0	271	86.3	0.11

^aMcNemar matched-pair chi-square (22).

^b $p < 0.05$.

were endorsed by less than 20% of the study group at both times 1 and 2. Student responses demonstrate that more PTSD-like symptoms were reported before December 3 than afterward. At initial interview, students endorsed a mean of 5.6 symptoms (range=0–12), while at follow-up, students endorsed a mean of 4.7 symptoms (range=0–11). Furthermore, a significant interaction between time and developmental stage of the child was found (repeated-measures multiple analysis of variance: $F=4.02$, $df=1$, 312, $p < 0.05$). Scheffé post hoc analyses indicated higher anticipatory stress index levels for elementary students than for high school students at both time 1 ($F=86.46$, $df=1$, 312, $p < 0.01$) and time 2 ($F=147.31$, $df=1$, 312, $p < 0.01$), as well as a

sharper decline in anticipatory stress index between the two interviews for high school students.

Mediating Factors

Finally, a group of three fully crossed, fixed factors, repeated measure design (age, time, mediating factor) analyses were run to determine factors related to the amount of anticipatory stress displayed, as well as changes from time 1 to time 2. Individual preparations for an earthquake were explored; there was a significant interaction only between planning and developmental level ($F=7.26$, $df=1$, 309, $p<0.01$). Elementary students who developed individual plans for dealing with an earthquake endorsed significantly more stress symptoms than elementary students without a plan ($F=6.78$, $df=1$, 309, $p<0.01$). Individual planning did not affect the stress response reported by high school students. On the other hand, family preparedness for earthquake was significantly associated with higher scores on the stress index for both developmental groups at both times ($F=6.39$, $df=1$, 302, $p<0.05$).

A second mediating factor studied involved actually doing something different during the week of December 3 because of the earthquake prediction. Students who reported doing something different had significantly higher anticipatory stress index scores than those who did nothing different ($F=17.97$, $df=1$, 310, $p<0.01$). Family response during the week was also studied. Elementary students seemed unaffected by family actions during the week. However, high school students who reported that their family did something different to contend with an earthquake reported more stress symptoms at time 1 than did high school students whose family did nothing ($F=4.77$, $df=1$, 310, $p<0.05$), and they demonstrated a significant decrease in symptoms between time 1 and time 2 ($F=10.07$, $df=1$, 310, $p<0.01$).

Talking about the earthquake prediction was also associated with anticipatory stress, with a significant interaction between talking and time ($F=6.87$, $df=1$, 310, $p<0.01$). The anxiety levels of those who did not talk to anyone about the earthquake predictions remained constant over time. However, those students who reported spending time talking about the possibility of an earthquake showed significantly higher levels of anticipatory stress at time 1 than those who did not ($F=7.79$, $df=1$, 310, $p<0.01$). Furthermore, post hoc analyses indicated a significant decrease in stress symptoms reported between time 1 and time 2 by those who talked about the prediction ($F=16.96$, $df=1$, 310, $p<0.000$). Thus, by time 2, both groups exhibited similar stress levels.

Two powerful variables associated with level of anticipatory stress were beliefs regarding the continued threat of an earthquake along the New Madrid fault and the timing of a future earthquake. Students who believed that an earthquake was going to occur as predicted had higher anticipatory stress index scores than those who did not; the scores of students who were not

sure fell in between ($F=6.26$, $df=2$, 308, $p<0.01$). After the earthquake did not occur, the highest levels of stress were exhibited by children and adolescents who continued to feel an imminent danger from earthquakes (i.e., they expected an earthquake within 1 year) and by those who did not believe that there would ever be an earthquake along the New Madrid fault, as opposed to those who thought that an earthquake would occur sometime in the future (more than 1 year) ($F=10.13$, $df=2$, 282, $p<0.01$).

DISCUSSION

The warning stage of a disaster, with ongoing states of expectation, has not been adequately studied in children, and the current study provides insight into how children cope with uncertainty and anticipation. This study documents the existence of a mild but prevalent PTSD-like reaction that arose from exposure to predictions of disaster. Duration of this stress reaction appears to be brief and, not surprisingly, highly associated with perceptions of continued threat.

In anticipation of an earthquake, many subjects reported elevated stress-related symptoms. Symptoms associated with this reaction were increased levels of anxiety including sleep problems, nightmares, trauma-related and generalized fears, and concerns about personal safety and safety of attachment figures. The four most commonly reported symptoms included thinking about an earthquake, concerns regarding destruction of homes and schools, and somatic complaints. Children and adolescents also reported subtle attitudinal changes regarding life expectancy and satisfaction regarding their community.

The data also indicate individual differences in stress response to the earthquake prediction, with some children and adolescents significantly more affected than others and with different reactions related to developmental level. Specifically, results of this study reveal that elementary students reported generally higher stress levels, which responded gradually to the diminished perceptions of threat. Those most highly symptomatic in anticipation of an earthquake seemed disproportionately anxious; they experienced earthquake-specific nightmares and fears and were concerned with their own welfare, that of their parents, and their home and school. The significance of concerns about personal safety and property in the development of stress-related disorders is consistent with work done by Shore et al. (15), who used the death of a family member and major property damage as indices of level of exposure.

Even without the actual experience of trauma, some children demonstrated enactments through playing earthquake-related games. Most of the games involved practicing what to do in the event of an earthquake or pretending to leave town for safer destinations. One made-up game, titled *Beat the Quake*, involved stomping feet. This game combined simulation of earthquake sensations and efforts to conquer it.

Through daily media coverage and community disaster preparations, the earthquake prediction for the New Madrid fault grew into a tale of traumatic proportions. Although the children and adolescents in the community did not experience an earthquake, they did perceive danger and react to those perceptions. One important phenomenon demonstrated is the substantial widening of the zone of danger to include those who only anticipate trauma. Indeed, operationalizing the definition of trauma is a consistent problem in the development of specific criteria for PTSD. Attempts to delimit the concept (DSM-III and DSM-III-R) provide subjective definitions of traumatic experience and raise significant diagnostic and research concerns (18, 23). An important issue to be addressed is whether exposure to a predicted disaster, in this case the warning stage for a major earthquake, can constitute trauma.

It is hoped that the examination of children exposed to predictions of trauma will give direction to development of methods to provide support to children during periods of anticipated trauma. Findings of higher stress levels in youngsters involved in disaster preparations and follow-through are contrary to the a priori hypothesis and are further complicated by developmental differences. Involvement in planning, theorized as an indication of internal locus of control, should mitigate reactions to trauma. Perhaps in the aftermath of an actual earthquake, involvement in preparations would be adaptive and help mitigate the development of PTSD; however, it is apparently associated with higher levels of anticipatory stress. This finding presents a curious dilemma for those who are interested in preventing or controlling anticipatory stress, yet realize the importance of adequate preparation.

Talking about the earthquake prediction, used as a measure of social support, was significantly associated with anticipatory stress reaction but in a direction opposite to that hypothesized. The explanation for this discrepancy may again indicate the differences in stress before disaster versus the optimal stress necessary for preparedness and adaptation. Before disaster, talking about the possibility of an earthquake may simply serve as a reminder of the predicted event. If anticipatory stress is viewed as an extension of PTSD, this reminder may only raise additional concerns about the expected trauma, rather than relieve them. After the impact stage, however, talking about the predicted disaster may help children feel better able to deal with the hardships that follow.

Although the current project was not designed to test the relationship between parental or adult anxiety and that of children, this relationship may provide an alternative explanation for the results presented. Talking about the earthquake and family plans for earthquake preparedness were significantly associated with higher levels of anxiety in the children and adolescents interviewed, a finding supportive of the earlier studies from World War II. Again, findings from the present study highlight differences in susceptibility to anticipatory stress according to developmental stage of the child.

Thus, in the development of disaster preparation programs, a key ingredient is identifying and supporting adaptive interactions between children and adolescents and their significant adults. Further study of this issue, with data collected directly from parents and other significant adults (i.e., teachers), would provide more conclusive findings.

Obviously, the symptom picture depicted in the present study is limited in scope by the hypotheses used in designing the interviews. In addition, some symptoms evidenced in the study group may be specific to the threat of an earthquake. In other words, the specific symptoms associated with anticipatory stress may be disaster-specific. Limitations of the current study include the absence of a comparison group of unexposed children and the lack of data collected directly from parents. Further study of an anticipatory stress response is necessary in order to clearly define its clinical characteristics and course.

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Obsessions in Obsessive-Compulsive Disorder With and Without Gilles de la Tourette's Syndrome

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***Objective and Method:** Although obsessive-compulsive disorder commonly occurs in many patients with Gilles de la Tourette's syndrome, little is known about the obsessions and compulsions of Tourette's syndrome and whether they differ from those seen in pure obsessive-compulsive disorder. The authors prospectively studied 10 subjects with obsessive-compulsive disorder and 15 subjects with obsessive-compulsive disorder and comorbid Tourette's syndrome by using the Yale-Brown Obsessive Compulsive Scale, the Leyton Obsessional Inventory, and a new questionnaire designed to emphasize the differences in symptoms between these two groups.*

***Results:** Subjects with comorbid obsessive-compulsive disorder and Tourette's syndrome had significantly more violent, sexual, and symmetrical obsessions and more touching, blinking, counting, and self-damaging compulsions. The group with obsessive-compulsive disorder alone had more obsessions concerning dirt or germs and more cleaning compulsions. The subjects who had both disorders reported that their compulsions arose spontaneously, whereas the subjects with obsessive-compulsive disorder alone reported that their compulsions were frequently preceded by cognitions. **Conclusions:** There are phenomenologic differences between obsessive-compulsive disorder and obsessive-compulsive disorder with comorbid Tourette's syndrome that may reflect differential involvement of neurochemical and neuroanatomic pathways.*

(Am J Psychiatry 1993; 150:93-97)

Georges Gilles de la Tourette first noted the association between obsessions and compulsions and the syndrome that bears his name in his original 1885 description (1). Since then, evidence on the links between obsessive-compulsive disorder and Tourette's syndrome has been strengthened. Family studies have shown that patients with either obsessive-compulsive disorder or Tourette's syndrome have a significantly greater number of family members with the other disorder (2). There is a high concordance for these two disorders among monozygotic twins (3). In addition, many patients with Tourette's syndrome have obsessions and compulsions (4, 5), and patients with obsessive-compulsive disorder frequently have tics (6). Some authors have suggested that

there may be a common gene which has as its phenotype either obsessive-compulsive disorder or Tourette's syndrome (7) or Tourette's syndrome and other psychiatric illnesses (8). Despite these interesting links, few studies have systematically compared the phenomenology of the obsessions and compulsions occurring in Tourette's syndrome and those occurring in pure obsessive-compulsive disorder. Some writers have argued that obsessive-compulsive symptoms in Tourette's syndrome roughly parallel pure obsessive-compulsive disorder (6); however, others have noted differences (9, 10). To investigate this issue we carried out the following prospective study.

METHOD

Ten subjects with obsessive-compulsive disorder and 15 subjects with obsessive-compulsive disorder and comorbid Tourette's syndrome were recruited from the outpatient clinic at a hospital for neurological disorders in London and enrolled in the study. All gave informed consent before the study began. Subjects were interviewed before they entered medication treatment trials.

Screening measures included a detailed history and physical examination, EEG, a single photon emission computed tomography (SPECT) scan, and serologic tests

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TABLE 1. Data on Subjects With Obsessive-Compulsive Disorder With and Without Tourette's Syndrome

Group	Age (years)		Sex ^a		Yale-Brown Obsessive Compulsive Scale Score		Leyton Obsessional Inventory Score	
	Mean	SD	Female	Male	Mean	SD	Mean	SD
Obsessive-compulsive disorder (N=10)	32.4	2.6	6	4	22.2	6.8	41.8	13.3
Obsessive-compulsive disorder and Tourette's syndrome (N=15)	33.2	11.1	4	11	24.8	4.9	31.5	12.5

^aSignificant difference between groups ($\chi^2=1.56$, $df=1$, $p<0.05$).

TABLE 2. Phenomenology of Obsessive-Compulsive Disorder in Two Groups of Subjects According to Results on the Yale-Brown Obsessive Compulsive Scale

Symptom	Group With Obsessive-Compulsive Disorder (N=10)		Group With Obsessive-Compulsive Disorder and Tourette's Syndrome (N=15)		χ^2 ($df=1$)	p
	N	%	N	%		
More common in obsessive-compulsive disorder						
Concern with bodily wastes or secretions	5	50	2	13	4.00	<0.05
Concern with dirt or germs	3	30	0	0	5.11	<0.03
Concern with environmental contamination	5	50	1	7	6.17	<0.02
Fear of not saying the right thing	4	40	1	7	4.16	<0.05
Dysmorphophobia	5	50	2	13	4.00	<0.05
Cleaning compulsions	3	30	0	0	5.11	<0.03
More common in obsessive-compulsive disorder with Tourette's syndrome						
Obsession with need for symmetry accompanied by magical thinking	1	10	9	60	6.25	<0.02
Fear of doing something embarrassing	1	10	7	47	3.70	<0.06
Fear of blurting out an obscenity	0	0	5	33	4.16	<0.05
Intrusive violent images	6	60	14	93	4.16	<0.05
Intrusive sexual thoughts	1	10	8	53	4.89	<0.03
Touching compulsion	1	10	14	93	11.78	<0.001
Rituals involving blinking or staring	0	0	8	53	7.84	<0.005
Self-injurious compulsions	0	0	10	67	11.11	<0.001
Hoarding	1	10	7	47	3.70	<0.06
Counting (arithmomania)	3	30	11	73	4.57	<0.04

to exclude secondary causes of Tourette's syndrome or obsessive-compulsive disorder. Exclusionary criteria included hepatic, renal, and cardiac disease, pregnancy, seizure disorder, mental retardation, and the presence of depression or significant psychiatric illnesses other than obsessive-compulsive disorder and Tourette's syndrome. Subjects were evaluated by the authors and given a detailed neurologic examination to assess for tics; they met the DSM-III-R criteria for obsessive-compulsive disorder or Tourette's disorder. Two of the 10 subjects with obsessive-compulsive disorder were also noted to have tics.

Subjects were administered the 72-item Yale-Brown Obsessive Compulsive Scale (11, 12), the questionnaire form of the Leyton Obsessional Inventory (13, 14), and a new questionnaire designed by us after noting clinical differences and interviewing patients with comorbid Tourette's syndrome and obsessive-compulsive disorder who were not included in this study (appendix 1).

Group means of parametric variables such as age and

Yale-Brown and Leyton scale scores were compared by using two-tailed Student's *t* tests. Group differences on categorical variables were compared by using chi-square analysis.

RESULTS

The two groups did not differ significantly in age or severity of disorder as measured by the Yale-Brown Obsessive Compulsive Scale and the Leyton Obsessional Inventory (table 1). The group with comorbid obsessive-compulsive disorder and Tourette's syndrome had a significantly higher percentage of men, in accordance with the known distribution of Tourette's syndrome.

Data from the Yale-Brown Obsessive Compulsive Scale (table 2) revealed that the group with obsessive-compulsive disorder had more frequent obsessions concerning bodily wastes and secretions, dirt or germs, and

TABLE 3. Phenomenology of Obsessive-Compulsive Disorder in Two Groups of Subjects According to Results on a Questionnaire for Distinguishing Obsessive-Compulsive Disorder From Obsessive-Compulsive Disorder With Tourette's Syndrome

Symptom	Group With Obsessive-Compulsive Disorder (N=10)		Group With Obsessive-Compulsive Disorder and Tourette's Syndrome (N=15)		χ^2 (df=1)	p
	N	%	N	%		
More common in obsessive-compulsive disorder						
A thought precedes the compulsion	9	90	1	7	17.36	<0.0001
More common in obsessive-compulsive disorder with Tourette's syndrome						
Compulsions arise from nowhere	2	20	15	100	17.64	<0.0001
Worry about blurting out an obscenity	0	0	9	60	6.95	<0.0001
Bodily harm	1	10	9	60	6.25	<0.02
Overawareness of bodily sensations	2	20	13	87	11.11	<0.001
Intrusive violent scenes	4	40	14	93	8.46	<0.003
Dirty words or thoughts	1	10	10	67	7.81	<0.005
Need to imitate others	0	0	9	60	9.37	<0.002
Sudden urge to destroy things	0	0	8	53	7.84	<0.005
Urge to offend others	0	0	5	33	4.16	<0.05
Profound urge to explore environment	2	20	10	67	5.23	<0.03
Impulse to hurt self	0	0	8	53	7.84	<0.005
Urge to touch self	2	20	9	60	3.89	<0.05
Urge to touch things	2	20	11	73	6.83	<0.008

environmental contamination, greater fear of not saying the right thing, greater dysmorphophobia, and more cleaning compulsions. The group with obsessive-compulsive disorder and Tourette's syndrome had more obsessions involving violent images, sexual thoughts, need for symmetry accompanied by magical thinking, and fear of blurting out an obscenity or doing something embarrassing. They also had higher rates of compulsions involving self-injury, blinking or staring, hoarding, touching, and counting.

The new questionnaire (table 3 and appendix 1) confirmed and expanded on the findings from the Yale-Brown Obsessive Compulsive Scale. The group with obsessive-compulsive disorder and Tourette's syndrome had more obsessions involving worrying about blurting out an obscenity, violent scenes, and obscene thoughts. This group also had more compulsions involving bodily harm and urges to imitate others, destroy things, offend others, explore the environment, injure themselves, touch themselves, and touch things. They also felt that they were overly aware of bodily sensations.

The two groups differed in their response to questions concerning the cause of or precedent for their compulsions. The group with obsessive-compulsive disorder responded that a thought often preceded their compulsions, whereas the group with obsessive-compulsive disorder and Tourette's syndrome responded that their compulsions arose spontaneously.

DISCUSSION

Some caution should be used in interpreting these results. Because of the rigorous screening measures, the

number of subjects involved was relatively small, which limits statistical power. The two groups differed significantly in sex ratio, reflecting the known differences in the incidence of these disorders in men and women. Our results are unlikely to have stemmed from this difference in sexual ratio, however, as a subanalysis of the female subjects with obsessive-compulsive disorder and Tourette's syndrome showed that they answered in a fashion similar to that of their male counterparts.

We considered whether the subjects in this study, and thus the results, may in some way reflect a selection and referral bias. The subjects with comorbid Tourette's syndrome were obtained from a hospital that is a national referral center for patients with that syndrome, whereas most subjects with obsessive-compulsive disorder alone were obtained from the London area. However, the two groups did not differ in severity of disorder, at least according to the Yale-Brown Obsessive Compulsive Scale and the Leyton Obsessional Inventory.

Finally, this study highlights the problem of distinguishing between a tic, a compulsion, and an obsession (15). For the purposes of this study, compulsions were defined as purposeful, complex motor acts. Tics were defined as meaningless, rapid, repetitive motor acts. Some persons would argue that compulsions in Tourette's syndrome are really complex motor tics. The distinction can be consistently made clinically, even if it is still debatable theoretically, by emphasizing the meaningfulness, rapidity, and repetitive nature of tics.

Despite these caveats, significant and interesting differences between the two groups emerged. The subjects with comorbid obsessive-compulsive disorder and Tourette's syndrome had significantly more violent and

sexual obsessions and had compulsions involving self-injury and touching. Some of these aspects of Tourette's syndrome have been previously described (4, 16, 17), but not the nature of the differences between Tourette's syndrome and obsessive-compulsive disorder, as we have done here. Interestingly, Holzer et al. (10) recently studied the phenomenology of obsessive-compulsive symptoms in obsessive-compulsive disorder sufferers with and without tics. Their group with obsessive-compulsive disorder and tics had significantly more obsessions and compulsions involving numbers, checking, repeating, touching, blinking, and staring and fewer compulsions involving cleaning and contamination. Thus, their group with obsessive-compulsive disorder and tics was similar to our group with obsessive-compulsive disorder and Tourette's syndrome.

The fear of blurting out an obscenity or doing something else embarrassing may be grounded in the fact that some patients with Tourette's syndrome have coprolalia or copropraxia. The obsessional need for symmetry with magical thinking is often seen clinically in these patients and has been described before (10). In contrast, the group with obsessive-compulsive disorder alone had significantly more obsessions and compulsions concerning the themes of contamination and cleanliness—themes virtually absent from the subjects with obsessive-compulsive disorder and Tourette's syndrome.

The questionnaire highlighted several additional differences between the two groups. The compulsion to imitate others can be viewed as an extension of the entire gamut of echo phenomena in Tourette's syndrome (echolalia, echopraxia, etc.). Similarly, the sudden urge in persons with Tourette's disorder to explore the environment has been described by others (9, 18, 19). The overawareness of bodily sensations in the subjects with comorbid obsessive-compulsive disorder and Tourette's syndrome may reflect the phenomenon of sensory tics (20–23). Importantly, the two groups differed significantly in terms of the antecedents of their compulsions: the group with both disorders largely reported that their compulsions arose de novo, and the group with obsessive-compulsive disorder alone reported that their compulsions were often preceded by a cognitive stimulus (guilt, worry).

These results become more interesting in light of recent genetic, neuroanatomic, and neuropharmacologic findings in these two disorders. Many researchers now feel that obsessive-compulsive disorder, obsessive-compulsive disorder with tics, and Tourette's syndrome represent different phenotypic expressions of the same underlying gene (2, 24–26). The differing phenomenology of the symptoms of obsessive-compulsive disorder may reflect underlying neurobiological differences within the obsessive-compulsive disorder spectrum. Recent advances in functional neuroimaging with positron emission tomography (27) and SPECT (28) may shed light on the differing neuroanatomic and neurochemical involvement in these disorders. Baxter (29) and Modell et al. (30) have proposed neuro-

anatomic models in which differential involvement of the frontal cortex (increased in obsessive-compulsive disorder) versus the basal ganglia (increased in Tourette's syndrome) reflect the clinical expression of obsessive-compulsive disorder, obsessive-compulsive disorder with tics, and obsessive-compulsive disorder with Tourette's syndrome. From this perspective, the difference in cognition preceding compulsions between the subjects with obsessive-compulsive disorder alone and the subjects with the two disorders may be important. One might speculate that in the subjects with obsessive-compulsive disorder and Tourette's syndrome, the compulsion arises de novo, or before a conscious thought, and thus arises from a subcortical trigger, possibly from the basal ganglia or thalamus. For the subjects with obsessive-compulsive disorder, in which it is known that there is orbital-frontal hyperactivity (31–33), the stimulus starts as a cognition arising from the frontal lobe, which then triggers a compulsion. More functional neuroimaging studies are needed to investigate these mechanisms.

At present, the limited treatment studies available show that obsessive-compulsive symptoms in Tourette's syndrome respond similarly to those in pure obsessive-compulsive disorder (7, 34), although there is some evidence that subjects with obsessive-compulsive disorder and tics and subjects with obsessive-compulsive disorder and Tourette's syndrome may preferentially respond to treatment with dopamine blockers (35–37). Further studies are needed to explore these differences in obsessive-compulsive disorder phenomenology and the underlying neurobiology and neuropharmacology.

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APPENDIX 1. Questionnaire for Distinguishing Obsessive-Compulsive Disorder From Obsessive-Compulsive Disorder With Tourette's Syndrome

1. Do you have urges to touch things?
2. Do you sometimes suddenly feel the urge to touch yourself or your bodily parts?
3. Do you have impulses to hurt yourself?
4. If you answered yes to the above question, is it because of feelings of guilt or uncleanness?
5. Do you sometimes have to repeat things several times, for no reason?
6. Do you sometimes have a profound urge to explore things in your environment?
7. Do you have the sudden urge to destroy things?
8. If you have sudden urges of any kind, are they preceded by thoughts of guilt or uncleanness?
9. When you act on your urges, do you feel unclean or guilty?
10. Do you sometimes have the urge to offend others?
11. Does a thought or motive come before your compulsions or urges?
12. Do your compulsions or urges arise from out of nowhere?
13. Do you feel a need to imitate other people?
14. Do dirty words or thoughts come into your head when you are thinking about other things?
15. Do bloody or violent scenes pop into your head when you are thinking about other things?
16. Are you overly aware of sensations from various parts of your body, even when you are thinking about other things?
17. Do you feel a need to do other things that you know will cause you bodily harm, such as touching hot objects or hitting yourself?
18. Have you worried about blurting out an obscenity or doing something sexual (like exposing yourself) in public?

Premonitory Urges in Tourette's Syndrome

James F. Leckman, M.D., David E. Walker, B.A., and Donald J. Cohen, M.D.

***Objective:** Tourette's syndrome traditionally has been viewed as a hyperkinetic movement disorder characterized by involuntary motor and phonic tics. Many patients, however, describe their tics as a voluntary response to premonitory urges. This cross-sectional study evaluated premonitory urges and related phenomena in subjects with tic disorders. **Method:** A total of 135 subjects with tic disorders, aged 8 to 71 years, completed a questionnaire concerning their current and past tic symptoms. Subjects were asked to describe and, if possible, localize their premonitory urges. The Yale Global Tic Severity Scale was used to assess current tic severity. The method of case finding does not provide prevalence data for premonitory urges. **Results:** Ninety-three percent of the subjects reported premonitory urges. Anatomical regions with the greatest density of urges were the palms, shoulders, midline abdomen, and throat. Eighty-four percent of the subjects reported that tics were associated with a feeling of relief. A substantial majority (92%) also indicated that their tics were either fully or partially a voluntary response to the premonitory urges. **Conclusions:** While epidemiological studies of tic disorders have yet to incorporate questions concerning premonitory urges, these results suggest that such urges may be commonplace in adolescent and adult subjects with tic disorders. These results challenge the conventional wisdom that tic behaviors are wholly involuntary in character. They also implicate brain regions involved in the processing of sensorimotor information in the pathobiology of tic disorders.*

(Am J Psychiatry 1993; 150:98-102)

Tourette's syndrome has been viewed traditionally as a hyperkinetic movement disorder characterized by involuntary motor and phonic tics (1). In 1980, Bliss (2) described his personal experience of the sensory phenomena that precede, accompany, and follow tics and challenged the conventional wisdom that tics are involuntary in character. Subsequently, Shapiro and colleagues (3) introduced the term "sensory tics" to describe some of these phenomena and reported that sensory tics alone or in combination with motor or phonic

tics were present in 8.5% of a large series of Tourette's syndrome patients. More recently, larger case series have been reported that have focused on either the nature of the sensory phenomena or their voluntary or involuntary character (4, 5). These reports suggest that premonitory sensory urges are frequently encountered in referral samples (4) and that most individuals do not experience tics as wholly involuntary (5). This paper adds to the growing literature by presenting data from 135 subjects with tic disorder who provided extensive data concerning premonitory urges and related mental phenomena.

METHOD

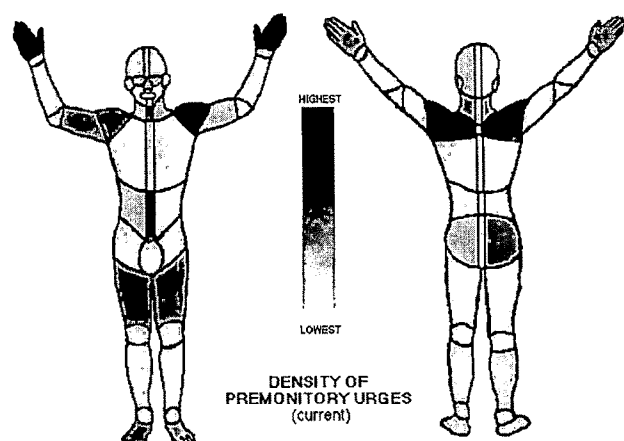
On the basis of numerous interviews with patients and a review of the available literature, a questionnaire (available on request from the first author) was developed to gather information on the onset, frequency, anatomical location, and character of premonitory sensory and related mental phenomena in tic disorder patients. Subjects responded to such questions as whether they had ever had or currently had premonitory tic sensations, whether these sensations were more mental or more physical in nature, the age at which they first became aware of these sensations, where the sensations

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FIGURE 1. Density of Current Premonitory Urges in 101 Subjects With Tic Disorders^a

^aThe densities of each anatomical region for the 101 respondents who completed the body figures were tallied, providing total densities for each region. The highest density on the scale represents 0.40 total premonitory urges per region per person; the lowest, 0 urges per region per person; and the midpoint, 0.20 urges per region per person.

were felt on the body, whether they helped to suppress tics, and whether they were affected by medications or certain life situations.

Subjects were instructed to rate the frequency of premonitory urges for eight common motor and phonic tics. Full-page body figures (front and back) were included in the questionnaire so that subjects could mark precisely the location of their premonitory urges. The location of premonitory urges was marked with an x , and the x was circled if relief was felt after the tic. In order to aid in the analysis, the body figures were subdivided into 87 separate surface regions, front and back, right and left, in accordance with the vernacular descriptions of body parts, e.g., right palm, left shoulder blade, and anterior right knee (figure 1). The right side was clearly delineated from the left by a series of midline regions running from the top of the head to the groin. In each of the 87 regions, the circled and uncircled x 's were tallied, providing a density for each anatomical region.

Subjects were asked about their perception of their tics—whether tics occurred without any warning and whether they considered the tics to be involuntary, voluntary, or a mixture of both. In addition, subjects were asked to complete the self-report versions of the Yale Global Tic Severity Scale (6) and the Yale-Brown Obsessive Compulsive Scale (7, 8). On the basis of our pilot data, adolescent and adult patients required 30 to 60 minutes to complete the questionnaire. Prepubertal subjects with parental assistance required somewhat less time.

Once the development of the instrument was complete, 36 questionnaires were distributed to patients at a tic disorder clinic of a university child study center. As

TABLE 1. Demographic and Clinical Characteristics of Subjects With Tic Disorders

Item	Clinic Subjects (N=36)	Nonclinic Subjects (N=99)	Total (N=135)
Sex			
Men	25	80	105
Women	11	19	30
Age (years) ^a			
Mean	29.5	31.6	31.0
SD	9.7	15.7	14.3
Range	13.8–51.3	8.8–71.6	8.8–71.6
Age at tic onset (years) ^b			
Mean	7.3	6.9	7.0
SD	3.3	2.6	2.8
Range	3.0–16.0	2.0–15.0	2.0–16.0
Yale Global Tic Severity Scale score ^c			
Mean	27.2	25.0	25.6
SD	8.1	8.5	8.4
Range	8.0–48.5	0–47.0	0–48.5
Yale-Brown Obsessive Compulsive Scale score ^d			
Mean	12.7	10.4	10.9
SD	8.5	7.1	7.4
Range	0–30.0	0–27.0	0–30.0

^aN=98 for nonclinic subjects and 134 for total.

^bN=96 for nonclinic subjects and 132 for total.

^cSum of motor and phonic tic scores (6). N=90 for nonclinic subjects and 126 for total.

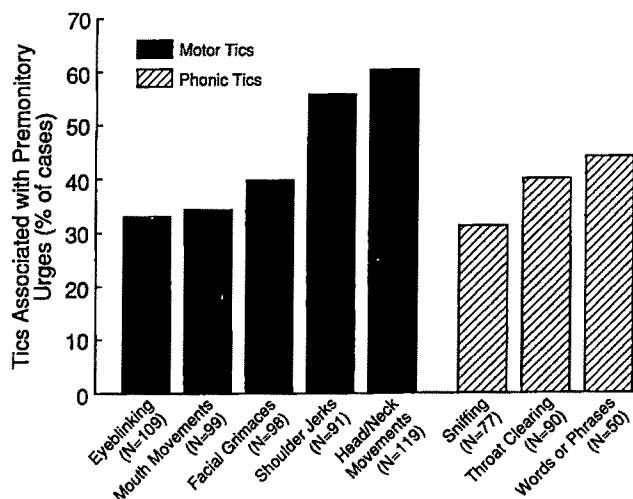
^dN=33 for clinic subjects, 81 for nonclinic subjects, and 114 for total.

in the pilot phase, the questionnaires were subsequently reviewed by clinicians familiar with the patient's case history and medical records for completeness, clarity, and accuracy. If questions arose during the review, the patients were contacted by telephone to obtain clarification and additional information.

With the approval of the Medical Committee of the Tourette Syndrome Association, 327 questionnaires were then mailed to tic disorder subjects in Connecticut, Rhode Island, Massachusetts, New York, New Jersey, Pennsylvania, and Illinois. Subjects were encouraged to contact the center if they encountered any difficulty in completing the questionnaire. A total of 99 questionnaires were returned from this distribution. All questionnaires were reviewed for completeness. Approximately 20% of the subjects who responded by mail were given follow-up telephone calls in order to clarify ambiguous responses or to complete unanswered questions.

No differences were observed between the clinic and nonclinic groups with regard to gender, current age, age at onset of tics, or current severity of either tics (Yale Global Tic Severity Scale) or obsessive-compulsive symptoms (Yale-Brown Obsessive Compulsive Scale) (table 1). Subsequently, the data from the both groups were combined to yield a total group of 135 individuals. The age range of the group was 8–71 years.

Statistical comparisons were carried out with Student's *t* test, Mann-Whitney *U* test, chi-square analysis, and analysis of variance when appropriate. Class variables included group (clinic versus nonclinic) and gen-

FIGURE 2. Frequency of Premonitory Urges Before Common Motor and Phonic Tics in 135 Subjects With Tic Disorders^a

^aSubjects indicated whether they almost always, frequently, occasionally, or never experienced a premonitory urge before these commonly occurring tics. Percents are based on the combined "almost always" and "frequently" responses divided by the total number of subjects reporting those tics.

der. Severity of tics (based on the Yale Global Tic Severity Scale score), severity of obsessive-compulsive symptoms (based on the Yale-Brown Obsessive Compulsive Scale), and current age were entered in analyses as continuous variables.

RESULTS

Ninety-three percent of 132 respondents (N=123) identified having a sensation (mental or physical awareness) ("an urge," "a feeling," "an impulse," "a need") to experience a tic during the past week, and 95% of 129 subjects (N=123) reported ever having had them. Males were more likely to report premonitory urges than females (97%, N=103, versus 83%, N=29; $\chi^2=5.0$, $df=1$, $p<0.05$). The mean age at which respondents first became aware of the premonitory urges was 10.0 years (SD=6.2), which averaged 3.1 years (SD=5.7) after the onset of the tics.

Selected narrative descriptions of the premonitory tic phenomena are presented in appendix 1. When asked if the sensation or urge was more mental or physical in nature, 109 respondents (89%) said that it was either partly or wholly a physical experience.

Head, neck, and shoulder tics were reported as being most frequently preceded by premonitory urges (figure 2). A significant gender difference was found in the frequency of sensations or urges before tics of throat clearing; 67% (N=18) of the female subjects with throat clearing tics had premonitory phenomena, compared with 33% (N=72) of the male subjects ($\chi^2=6.67$, $df=1$, $p=0.01$).

According to the body figure data (N=101), the mean

number of anatomically distinct urges experienced during the previous week was 8.7 (SD=9.4, range=0-46). The anatomical regions with the highest density of premonitory urges were, in descending order, left palm, right shoulder blade, right palm, left shoulder, left shoulder blade, midline abdomen, throat, right shoulder, back of right hand, front of right thigh, front of right foot, back of left hand, inside of right upper arm/front of left thigh/left eye (last three items equal or in descending order), right eye (figure 1).

Thirty-nine subjects indicated that their urges were completely bilaterally symmetrical in location. Another 29 subjects indicated that half or more of their urges occurred in a bilaterally symmetrical pattern, and four subjects reported some (less than 50%) bilateral symmetry. Of the remaining 29 respondents with asymmetrical patterns, 19 (66%) were entirely unilateral; nine experienced urges only on the right side, and 10 had urges exclusively on the left side.

Most of these urges were judged to be felt in muscle. Forty percent of the respondents reported that the feeling was exclusively felt in the muscle, while another 24% felt it to be located in both muscle and joints. Just 8% felt it exclusively in their joints, and only 3% felt the sensation in their skin.

A total of 121 (92%) of 132 subjects reported that they experienced their tics to be partly or wholly voluntary. This finding was not affected by the severity of tic symptoms. Interestingly, 50% of the female subjects felt that their tics were completely voluntary, compared to only 28% of the male subjects ($\chi^2=5.60$, $df=1$, $p<0.05$). When asked if there were certain tics that always occurred without warning, 57 (48%) of 122 respondents reported none.

Medications and certain life situations affect the quality and frequency of pre-tic sensations and urges. According to 63 of 101 respondents, the sensations and urges were altered by medications. Many of these individuals mentioned that neuroleptics reduced the frequency and intensity of the premonitory urges. Moreover, 92 (77%) of 120 respondents noted that apart from medications, other situations or circumstances also affected their pre-tic urges and sensations; stress and anxiety increased the urge to tic, while relaxation and concentration decreased the urge.

The awareness of the premonitory urges can facilitate tic suppression, according to some of the respondents. Specifically, 24 individuals indicated that this awareness helped them to suppress their tics. The severity of tic symptoms was not a factor in this analysis.

Apart from the few gender differences noted earlier, univariate analyses using current age, tic severity, obsessive-compulsive symptom severity, and clinic versus non-clinic status did not yield statistically significant findings.

DISCUSSION

This cross-sectional survey of 135 subjects with tic disorder confirms Bliss's original observations (2) that

premonitory urges are commonplace among adolescents and adults with tic disorders and that subjects with tic disorders frequently experience their movements as being a voluntary response to these unwanted urges. These findings are consistent with those reported by Kurlan and associates (4), who found in a telephone survey that 74% of 35 patients with Tourette's syndrome reported having a sensation or feeling before tics. The data in the report by Lang (5), in which only 7% of 60 tic disorder patients experienced all tics as being involuntary, are virtually identical to the 8% figure found in our data. However, given the case finding procedures used in each of these studies, the age-specific prevalence of premonitory urges among tic disorder patients remains to be determined.

The premonitory urges are important clinical phenomena that frequently cause distress in their own right. Indeed, several of the patients in the present study spontaneously reported that the experience of these urges was more troublesome than their tic behaviors. In addition to being a source of constant distraction, the quasi-volitional nature of the urges was psychologically burdensome to some patients (9).

The reported lag of 3 years, on average, between the onset of tics and the initial awareness of premonitory urges is also intriguing. Does this reflect a maturational shift in the cognitive processing of sensory information so that these subtle and elusive phenomena can enter the individual's conscious awareness, or is the interval between tic onset and these cognitive/somatosensory phenomena more a function of the location and type of tics involved? The latter possibility is consistent with data concerning the mean age at onset for tics involving those anatomical regions most closely associated with premonitory urges. For example, Shapiro and associates (3) reported that the mean age at onset for tics involving the hands, shoulders, and abdomen is 11.4, 10.1, and 13.7 years, respectively, whereas the mean age at onset for motor tics in general is 6.7 years. These data also underscore that not all tics are associated with premonitory urges. For example, one patient was largely oblivious to a frequent eye blinking tic but was severely troubled by a shoulder jerk that he experienced as being prompted by a premonitory urge localized in a particular spot over his left scapula.

The frequent coupling of premonitory urges with tic behaviors is heuristically interesting in part because it resembles the coupling of obsessional thoughts or premonitory urges associated with the performance of compulsions or rituals by patients with obsessive-compulsive disorder. The extent to which these phenomena are governed by similar neurobiological mechanisms is unknown. Given the evidence for a common genetic vulnerability underlying both Tourette's syndrome and some forms of obsessive-compulsive disorder (10), it may be reasonable to speculate that the repetitive coupling of mental/sensory information and fragmentary behaviors reflects a deeper commonality of these disorders.

The basal ganglia and related cortical and thalamic structures have been implicated in the pathobiology of

Tourette's syndrome and obsessive-compulsive disorder (11) and may likely play a critical role in the occurrence of premonitory urges. Functionally, the basal ganglia are composed of pathways that contribute to the multiple parallel cortico-striato-thalamocortical circuits that concurrently subserve a wide variety of sensorimotor, motor, oculomotor, cognitive, and "limbic" processes (12, 13). It has been hypothesized that Tourette's syndrome and etiologically related forms of obsessive-compulsive disorder are associated with a failure to inhibit subsets of the cortico-striato-thalamocortical minicircuits (14). Specifically, the processing of somatotopically organized sensory information in parallel with adjacent circuits that process information associated with both the planning and performance of motor behaviors may provide the neuroanatomic basis for the premonitory urges of Tourette's syndrome and other tic disorders (15, 16). Although the neurobiological defect that underlies Tourette's syndrome and etiologically related conditions remains unknown, a more complete understanding of these disorders will likely illuminate mechanisms that regulate the activity of the multiple parallel cortico-striato-thalamocortical circuits that subserve much of the normal cognitive, behavioral, and emotive repertoire.

Future studies of patients with chronic tic disorder should include measures of the frequency, intensity, and interference associated with premonitory urges. It may also be important to explore the relationship between these sensorimotor phenomena and the perceptually driven need of some individuals with obsessive-compulsive disorder for things to look, feel, or sound "just right."

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APPENDIX 1. Descriptions of Premonitory Tic Phenomena by Patients With Tourette's Syndrome

"I feel that I have too much energy and have to get some of it out so I do that."

12-year-old boy

"like I have to hiccup but it won't come out."

13-year-old girl

"A need to tic is an intense feeling that unless I tic or twitch I feel as if I am going to burst. Unless I can physically tic, all of my mental thoughts center on ticking until I am able to let it out. It's a terrible urge that needs to be satisfied."

21-year-old woman

"A feeling of pressure—a need that's very hard to describe, like something itches deep inside you—but no place you can describe; and the only way you can relieve this need is by tics. It's like your brain itches, or your insides are being tickled . . ."

24-year-old man

"I guess it's sort of an aching feeling, in a limb or a body area, or else in my throat if it precedes a vocalization. If I don't relieve it, it either drives me crazy or begins to hurt (or both)—in that way it's both mental and physical."

27-year-old woman

"I always feel the urge prior to every tic. It's like an intense build-up of pressure that's relieved only by ticking."

43-year-old woman

"I have to 'do it one more time,' or 'complete something,' or make something symmetrical."

71-year-old man

Suicidal Behavior and Risk Factors Among Runaway Youths

Mary J. Rotheram-Borus, Ph.D.

Objective: The goal of this study was to describe suicide attempts and risk factors among runaway adolescents. **Method:** A structured interview format was used to assess suicidal behavior and suicide-related risk factors among a consecutive series of 576 predominantly black or Hispanic runaway adolescents at intake into four publicly funded runaway programs in New York City over a 2-year period. There were no significant differences in age, gender, race/ethnicity, education, or socioeconomic status among the adolescents at the four runaway program sites. **Results:** Thirty-seven percent of the youths had previously attempted suicide, and 44% of the attempters had made an attempt within the previous month. Females were significantly more likely than males to have attempted suicide and to be depressed. Male runaways were far more likely to have attempted suicide than nonrunaway male adolescents described in previously published reports. Runaways with histories of attempting suicide were significantly more likely to be currently suicidal and depressed. **Conclusions:** This study indicates the need for systematic screening of runaway adolescents for suicidal ideation at residential shelters for youths. (Am J Psychiatry 1993; 150:103–107)

Even though adolescent suicide has increased threefold in the last 25 years (10 per 100,000 deaths per year [1]), it remains a relatively rare and unpredictable event (2). Shaffer et al. (3) have argued, therefore, that researchers should focus on subgroups of potentially suicidal youths whose background places them at high risk. Adolescent runaways are one potentially high-risk group. In an epidemiological survey conducted over a 2-week period, Shaffer and Caton (4) found that 33% of female and 16% of male runaways had attempted suicide—rates higher than those reported from high school samples (e.g., 3% [5] and 7%–13% [6]), community samples (e.g., 2% [7] and 2.4% [8]), and adolescent clinic patients (e.g., 12.2% [9] and 34% [10]). In addition, the lives of runaways are characterized by high stress (11), a factor related to suicide among high school students (12).

Although the frequency of suicide attempts appears high among runaways, we know little about their suicidal acts and suicide-related risk factors. Information on suicidal behavior among minority youths is particularly lacking (9), especially among those in high-risk groups

such as runaways. Therefore, one goal of this study was to describe suicide attempts among runaways. Although there are substantial ethnic differences in the rates of suicide (1), there are few data on attempted suicides and suicide-related risk factors among minority adolescents considered at risk. This project examined suicidal behavior and suicide-related risk factors among runaways who were predominantly black or Hispanic.

We selected the suicidal risk factors of interest on the basis of a review of the literature. The following risk factors, based on statistical profiles of persons who had made successful suicide attempts, emerged: 1) male sex and white race (3); 2) current suicidal ideation and concrete plans to die (13); 3) past suicidal behavior—specifically, a previous suicide attempt, more than one previous attempt (because rates of reattempts range from 31% to 50%), and attempted suicide with a method other than ingestion of pills (14–19); 4) depression (3, 6, 8–10, 20–22); 5) conduct problems (8, 9, 23, 24); 6) drug and alcohol abuse (8, 10, 18, 25–27); and 7) suicidal behavior among peers and family (28, 29). Given the gender and ethnic differences found for persons who had committed suicide, we also examined these risk factors among attempters and nonattempters.

METHOD

A consecutive series of 260 male and 316 female adolescents presenting at four runaway programs in New York City were recruited at intake over a period of 2 years. No youth refused to participate in the study. Cooperation was atypically high because of positive relationships be-

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tween the staff members and the youths and the timing of the assessment, i.e., at the intake interview. The data were collected as part of a more extensive project for prevention of suicide attempts in shelters for runaways (30). These were all of the publicly funded shelters in Manhattan during the recruitment period. Three of the runaway programs were residential shelters. One residential program closed during the course of the study and another opened; therefore, the duration of recruitment varied across sites. Each residential shelter served predominantly minority inner-city youths, housing 20–25 males and females and admitting runaways 24 hours a day. The non-residential runaway program was located in the crisis unit of an agency providing comprehensive services for adolescents (i.e., health care and legal, educational, and recreational services). There was no control group for this study. However, the findings can be compared to previously reported prevalence estimates obtained from high schools as well as community-based and clinic samples (5, 7, 9).

There were no significant differences in age, gender, ethnicity, education, or socioeconomic status among the youths attending or recruited from any of the four programs. Of the 576 adolescents studied, 43% were black, 32% Hispanic (primarily Puerto Rican and Dominican), 11.5% non-Hispanic white, and the remainder biracial or members of other minority groups. Ninety-two percent lived in New York City. The mean age was 16.3 years (range=12–17). Forty-eight percent were enrolled in school, and 10th grade was the mean educational level. Although the definition of homelessness is controversial (31), 9.3% of the study group reported being thrown out of their homes, and most youths reported that problems at home were the major reason for leaving home. Seventeen percent of the subjects had run away five or more times.

Following approval by the Institutional Review Board of the New York State Psychiatric Institute, screening for suicidality was instituted as part of the intake procedure of each program. With informed and voluntary consent, a 10- to 25-minute interview regarding suicidality was administered by child care workers.

Before the study was implemented, prompt clinical care for current suicidality was ensured. 1) Staff members received 10 hours of training in assessing suicidality. 2) The reliability of the interviews was supported by intraclass correlations of 0.76 and higher on the ratings of three videotapes of adolescents of varying suicidality by staff members, agency supervisors, and the research team. Also, there was an increase in the assessment of staff members' knowledge about suicide from 63% before the training to over 90% afterward (30). 3) At each agency protocols were established for obtaining psychiatric evaluation of youths considered at high risk and for access to a network of services providing outpatient care and hospitalization.

To screen for suicidality and suicide-related factors (32), a structured interview was developed and pilot-tested with 158 youths over a 6-month period. Because the potential time for assessment was limited and the

staff members were not skilled clinicians, diagnostic assessments for depression, conduct problems, and substance abuse were not feasible. Instead, brief measures were developed and arbitrary criteria were set for assessing the presence or absence of suicide risk factors in six domains: current suicidality, history of suicidality, depression, conduct problems, alcohol and drug abuse, and family and peer suicide.

For the assessment of current suicidality, youths reported, for the past 2 weeks, the frequency of suicidal ideation (e.g., "thought about suicide one time," "was unable to think of anything else"), desiring to die, and having a plan to kill themselves. Suicidal ideation was significantly correlated with plans to die ($r=0.47$, $df=564$, $p<0.001$). Therefore, these two items were considered as one risk factor. Youths who reported daily suicidal ideation or a plan to kill themselves met the criterion for current suicidality.

For history of suicidality, we assessed the frequency of periods of serious ideation (every day for a week), desiring to die, and having plans for suicide, as well as the number of past suicide attempts (trying to hurt oneself with the intention of dying). Information elicited about past attempts included the timing of the most recent attempt, methods, circumstances (e.g., presence of others), precipitating events, history of suicide-related hospitalizations, use of alcohol and drugs surrounding an attempt, and counseling and therapy received that addressed suicidality. There were relatively high correlations between a history of a past attempt and having made more than one previous attempt ($r=0.51$, $df=457$, $p<0.05$), between a history of a past attempt and attempting suicide with a method other than ingestion of pills ($r=0.43$, $df=451$, $p<0.05$), and between more than one previous attempt and using a noningestion method ($r=0.47$, $df=495$, $p<0.05$).

Depression during the past week was assessed by means of a 21-item depression scale with symptoms adopted from the Schedule for Affective Disorders and Schizophrenia for School-Age Children, Present Episode (K-SADS-P) (33) and the Center for Epidemiologic Studies Depression Scale (34). This scale did not assess diagnosis or approximate a diagnosis. Sixteen problem symptoms were rated: lonely, bothered by daily events, fearful, not feeling as good as others, crying, restless sleep, poor appetite, felt blue, trouble concentrating, depressed, people unfriendly, activities an effort, could not "get going," felt disliked, talking less, and sad. For each item, youths reported whether the symptom occurred on less than 1 day (rated 0), 1–2 days (rated 1), 3–4 days (rated 2), or 5–7 days (rated 3). Three additional items (enjoyed yourself, happy, hopeful) with the same response scale were given a positive valence, and scores were reversed for these items. Two items indicating weight loss and hours slept were assessed on a 0–3 scale as above. Scores on all items were summed to yield one score, with a mean score added for missing items. On the basis of examination of distributions across items and consultation with clinicians, we set the criterion for depression as a score above the mean of 2.6 per item.

TABLE 1. Suicide-Related Risk Factors Reported by Adolescents Admitted to New York City Programs for Runaways

Risk Factor	Runaway Males			Runaway Females			Total		
	N	With Risk Factor		N	With Risk Factor		N	With Risk Factor	
		N	%		N	%		N	%
Current suicidal ideation	260	49	19	316	41	13	576	90	16
Current suicide plan	253	13	5	307	21	7	560	34	6
Past suicide attempt ^a	260	75	29	316	139	44	576	214	37
Made repeated attempts	72	20	28	116	42	36	188	62	33
Attempted suicide in the past month	72	33	46	116	49	42	188	82	44
Used a noningestion method	70	9	13	116	15	13	186	24	13
Depression ^b	260	114	44	316	196	62	576	310	54
Conduct problems ^c	260	126	48	316	86	27	576	212	37
Alcohol and drug abuse	260	47	18	316	50	16	576	97	17
Suicide among friends	260	18	7	316	26	8	576	44	8
Suicide in the family	260	21	8	316	38	12	576	59	10

^aSignificant difference between males and females ($\chi^2=4.8$, $df=1$, $p<0.02$).

^bSignificant difference between males and females ($\chi^2=14.1$, $df=1$, $p<0.0001$).

^cSignificant difference between males and females ($\chi^2=19.3$, $df=1$, $p<0.0001$).

The items from the conduct disorder scale of the K-SADS-P interview were used to assess behavior problems, excluding drug and alcohol abuse. These items did not assess a subject for the diagnosis of conduct disorder but were chosen to reflect a range of problems. There were two interview sections. The first section contained nine items (missed school, destroyed property, belonged to a gang, teased children, fought, had family trouble, lied, stole, set fires). These items were rated as occurring over the past 2 months not at all (rated 0) to four or more times per week (rated 3). In the second section, youths reported the frequency of arrests, running away, dropping out of school, and expulsion from school. For each item in this section, the actual number of occurrences was reported. Even though the rating scales in the two sections varied, the dimensions assessed were similar. Therefore, the summary score for conduct problems was calculated by summing the items for each section and standardizing for the study group (i.e., changing to z scores). Youths with a z score greater than 1.0 (one standard deviation from the group mean) were considered to have conduct problems.

Two questions assessed alcohol and drug use during the past 2 months, specifying the frequency of use from not at all (rated 0) to more than four times a week (rated 3). Arbitrarily, the criterion for the presence of alcohol and drug abuse was set at use more than twice a week.

For the factor of family and peer suicide, the numbers of peers (younger, same age, and older), nuclear family members, and extended family members who attempted or committed suicide were summed.

RESULTS

As shown in table 1, 37% ($N=214$) of the runaways had attempted suicide in the past, many in the month before they sought services at the runaway programs. Forty-six percent of these had attempted suicide once;

the mean number of attempts was similar for males (2.1, $SD=1.1$) and females (2.2, $SD=0.9$). Most attempts (62%) had occurred while the runaways were alone. Most attempters (64%) had ingested drugs; however, females were more likely than males to have ingested drugs (76% and 52%, respectively; $\chi^2=9.2$, $df=1$, $p<0.01$). Other methods used by the attempters included wrist cutting (11%), jumping (16%), reckless behavior (7%), using guns (5%), and hanging (5%). Trouble at home (22%), arguments (16%), disappointments (16%), and humiliations (10%) were the most common precipitating events, but attempts also followed trouble at school (5%), assaults (4%), and sexual abuse (4%). Pregnancy (<1%) and trouble with the law (<1%) were not common precipitants. About one-third of the attempters had been hospitalized (38%) and received therapy (35%) following their attempt, and 23% reported alcohol and drug use before the attempt.

Of the whole group of 576 runaways, about half (47%) reported never having a period of serious suicidal ideation (every day for a week), and 12% reported four or more periods of serious ideation. Ninety-four percent of those with three or more periods of suicidal ideation had attempted suicide. Those who reported serious ideation also reported intending to harm themselves (87%) and wanting to die (92%).

Chi-square analysis was used to examine ethnic and gender differences on each factor. There were no significant ethnic differences (between black and Hispanic subjects) in suicidal ideation, past suicidal behavior, or suicide-related risk factors. Compared to males, females reported significantly more past suicide attempts and depression and significantly fewer conduct problems (table 1).

In the previous 2 months, most depressive symptoms were reported to have occurred on 2–3 days of the last week on average (mean score=1.4, $SD=0.7$ [1=1–2 days a week; 2=3–4 days a week]). Reports of the average frequency of depressive symptoms were as follows: less than 1 day in the last week, 3.9%; 1–2 days in the last

week, 32.8%; 3–4 days, 33.4%; 5–7 days, 26.0%. Females reported significantly more crying ($F=28.1$, $df=1$, 438, $p<0.01$), fearfulness ($F=4.3$, $df=1$, 389, $p<0.05$), and difficulty in “getting going” ($F=5.6$, $df=1$, 419, $p<0.01$) than males.

The runaways reported conduct problems such as dropping out of school (44%), being truant (52%), being expelled from school (22%), getting arrested (14%), destroying property (10%), and belonging to a gang (8%). Fighting with peers (30%), fighting with younger children (8%), and trouble at home (57%) were also common. Among those who reported a conduct problem, the mean frequency score per week was 1.4 ($SD=1.7$) (1=1–2 days a week; 2=3–4 days a week).

Examining the overlap among suicide risk factors, we found that 46% of the subjects reported one risk factor, 36% had two or three risk factors, 16% had four or five, and 2% reported six or seven. There were neither gender nor ethnic differences in suicide-related risk factors. However, those who had attempted suicide in the past differed from the nonattempters in current suicidal ideation (26% and 10%, respectively; $\chi^2=17.9$, $df=1$, $p<0.001$), current plans for suicide (23% and 2%; $\chi^2=50.2$, $df=1$, $p<0.001$), and depression (89% and 38%; $\chi^2=43.6$, $df=1$, $p<0.001$). Of the seven risk factors, suicide attempters had significantly more (mean=3.1, range=0–7) than nonattempters (mean=1.2, range=0–6) ($F=289.5$, $df=1$, 457, $p<0.001$).

DISCUSSION AND CONCLUSIONS

The field sites placed a major limitation on data collection for this study. Following their initial training, child care workers in each agency were supervised by the administrators of the runaway programs. It was not possible for the research staff to monitor the quality of the staff members' assessments at each agency or to assess interrater reliability among interviewers across time and agencies. However, there are several indications that the child care workers were reliable interviewers: staff members' ratings matched supervisors' ratings when videotaped clinical interviews were viewed, and staff members' knowledge regarding adolescent suicide increased following training (30). The recruitment context also did not allow us to assess diagnoses and resulted in our setting arbitrary criteria for decisions on the presence or absence of suicide-related risk factors. Finally, there were relatively few non-Hispanic white adolescents in the study group, limiting ethnic comparisons to black and Hispanic youths.

Despite these limitations, the findings of this study were similar to those of Shaffer and Caton (4). The reports of past suicide attempts among the runaways were very numerous compared to prevalence estimates obtained in high schools, community-based samples, and clinic samples (5, 7, 9). More than one-third of the present study group had attempted suicide in the past, and many of these attempts were recent. Because it has often been found that suicide attempters reattempt sui-

cide and experience persistent psychiatric symptoms (35), these rates are disturbing and indicate that methods should be implemented to identify suicidal runaways. In fact, 30% of the runaway subjects who had attempted suicide had reattempted suicide. In addition, current suicidality was associated with a past attempt, and past attempters were currently more depressed than nonattempters.

Furthermore, the fact that many of these adolescents had attempted suicide in the month before they entered the runaway programs suggests that staff members in shelters for runaways must be trained to assess suicidality at intake. While less than 2% of runaways appear to require psychiatric hospitalization (30), our data indicate that many runaways need therapeutic intervention because of potential suicidality.

Runaway females were significantly more likely to have attempted suicide. However, in comparison to adolescent males in general, runaway males were at extremely high risk. Epidemiological surveys have found that the ratio of male to female suicide attempters is about 1:10 (1), but in this study the ratio was 0.8:1. Therefore, suicidal behavior and risk factors must be further investigated, particularly among runaway males, to identify factors in their lives that are associated with suicide attempts.

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Suicidal Behaviors in Adult Psychiatric Outpatients, I: Description and Prevalence

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Objective: Because the findings of previous studies of suicidal behaviors in psychiatric outpatients may not necessarily generalize to outpatients with a wide spectrum of psychiatric diagnoses, the authors evaluated the prevalence of suicidal behaviors in a large general psychiatric outpatient clinic whose patients represented a full spectrum of psychiatric illness. **Method:** A total of 651 patients participated in the study between 1987 and 1989. These patients had sought treatment at the outpatient psychiatry department of a private nonprofit hospital. Before being interviewed for treatment, all patients were given a comprehensive self-rating survey packet that included the Harkavy Asnis Suicide Survey and the Hopkins Symptom Checklist-90. The Harkavy Asnis Suicide Survey is a self-report questionnaire that assesses demographic variables, current and past history of suicidal behaviors of the patient as well as family members and peers, and a detailed description of each previous attempt. **Results:** Fifty-five percent of the patients had a history of suicidal ideation, and 25% reported at least one previous suicide attempt. Approximately half of the suicide attempters reported multiple attempts. The predominant methods of attempt were overdose (53%), jumping (17%), and wrist cutting (17%). Suicidal behavior was prevalent in most diagnostic groups. The rates of suicidal ideation among patients with mood disorders (major depression, dysthymia, and bipolar disorder), adjustment disorders, and alcohol/substance abuse were significantly greater than that of the patients with generalized anxiety disorder. **Conclusions:** The authors conclude that suicidal behavior is prevalent among patients who seek treatment in a general outpatient department.

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Most studies examining the prevalence of suicidal behaviors in psychiatric populations focus on suicidal behaviors within specific diagnostic groupings, e.g., mood disorders or schizophrenia (1-3). There is a paucity of investigations of suicidal behaviors in general psychiatric outpatient populations. Two major studies have evaluated prevalence rates of suicidal behaviors in outpatients. Linehan and Laffaw (4) reported a prevalence rate of 10% for a previous history of suicide attempts in a small clinic population (N=59); however, the nature of this psychiatric population, the patients' specific psychiatric diagnoses, and the specific questions asked were not clarified. Beck et al. (5) evaluated 900 outpatients referred to a cognitive therapy

clinic and found that 4% had a history of previous suicide attempts. They also found that the percentage of patients with mood disorders who had made previous attempts was significantly higher than the percentage of patients with panic disorder who had made previous attempts (7.0% versus 0.7%). Finally, Beck et al. found that patients with major depression had a higher rate of suicidal ideation, measured by the Scale for Suicidal Ideation and the Beck Hopelessness Scale, than patients with panic disorder or other psychiatric disorders. These two studies are striking because they suggest that suicidal behaviors in psychiatric outpatients are relatively rare and that suicide attempts tend to occur less frequently in panic disorder than in mood disorders.

These studies may be limited in that their findings do not necessarily generalize to outpatient settings where a wide spectrum of psychiatric diagnoses is represented. Therefore, we decided to evaluate the prevalence of suicidal behaviors in a large general psychiatric outpatient clinic. The patients in this study represent a full spectrum of psychiatric illness, which distinguishes this

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TABLE 1. Prevalence of Suicidal Behaviors in 651 Patients in a Psychiatric Outpatient Department

Diagnosis	Suicidal Ideation			Suicide Plan			Suicide Attempt		
	Total Patients Assessed	Patients Who Ever Had Ideation		Total Patients Assessed	Patients Who Ever Had Plan		Total Patients Assessed	Patients Who Ever Made Attempt	
		N	% ^a		N	%		N	% ^a
Overall	630	346	55	450	141	31	651	164	25
Major depression	228	146	64 _{a,b,c}	178	64	36	235	82	35 _{a,b}
Bipolar disorder	49	31	63 _{d,e}	37	14	38	52	16	31
Dysthymia	43	27	63 _{f,g}	35	8	23	44	13	30
Panic disorder	64	19	41 _c	29	5	17	47	4	9 _a
Generalized anxiety disorder	28	5	18 _{g,d,f,h,i}	13	4	31	30	5	17
Schizophrenia	67	32	48	44	18	41	69	20	29
Psychotic disorder not otherwise specified	34	17	50	24	6	25	35	5	14
Adjustment disorder	65	34	52 _h	42	9	21	68	7	10 _b
Organic mental disorder	18	4	22 _{b,c,g}	10	1	10	19	1	5
Alcohol and/or substance abuse	52	31	60 _i	38	12	32	52	11	21

^aPercentages sharing subscripts are significantly different at $p < 0.005$ (Bonferroni correction).

study from other prevalence studies of suicidal behaviors in outpatients.

METHOD

A total of 651 patients participated in this study between the years of 1987 and 1989; 254 (39%) of these patients were men and 397 (61%) were women. Their mean age was 39.7 years ($SD=15.2$). The racial distribution was 41% Hispanic ($N=264$), 31% white ($N=204$), 25% black ($N=163$), and 3% "other" ($N=20$). Thirty-five percent of the patients had previous inpatient hospitalizations.

The patients had sought treatment at the outpatient psychiatry department of a private nonprofit hospital. As part of the intake evaluation, all patients were given a comprehensive self-rating survey packet to complete before being interviewed. The self-rating packet included the Harkavy Asnis Suicide Survey demographic form (6) as well as the Hopkins Symptom Checklist-90 (7). The Harkavy Asnis Suicide Survey is a specific self-report questionnaire developed by the Department of Psychiatry at Albert Einstein College of Medicine to assess suicidal behaviors. It includes questions on demographic variables (e.g., age, sex, race, marital status), current and past history of suicidal behaviors (ideation, plan, attempt) of the patient as well as family members and peers, and details of each previous attempt (age of patient at time of attempt, method of attempt, communication of suicidal intent before and/or after the attempt, whether a subject wanted to die and/or expected to die, and whether a subject needed medical attention as a consequence of the attempt). Methods of attempt included hanging, shooting, drowning, jumping, overdosing, wrist cutting, and carbon monoxide poisoning (gas).

On completing these forms, each patient participated

in a semistructured intake interview that obtained information such as current and past psychiatric history and mental status, conducted by a trained mental health professional (social worker, psychologist, or psychiatrist). Subsequently, patients were given DSM-III-R diagnoses. On the basis of the primary DSM-III-R axis I diagnosis that patients were given at the intake interview, they were grouped into 10 diagnostic categories: major depression ($N=235$), bipolar disorder ($N=52$), dysthymia ($N=44$), panic disorder ($N=47$), generalized anxiety disorder ($N=30$), schizophrenia ($N=69$), psychotic disorder not otherwise specified ($N=35$), adjustment disorder ($N=68$), organic mental disorder ($N=19$), and alcohol and psychoactive substance abuse ($N=52$). Other diagnostic groups (e.g., obsessive-compulsive disorder, uncomplicated bereavement) are not represented in the sample because of their infrequent presentation in our outpatient department.

Chi-square tests were used to test for significant group differences when groups were compared on categorical variables (e.g., attempt versus nonattempt). Analysis of variance was used to compare groups on continuous variables such as age at first attempt. Due to multiple comparisons, significance levels were adjusted by using the Bonferroni correction.

RESULTS

The patients' histories of suicidal behaviors are summarized in table 1. Fifty-five percent of the patients reported ever having suicidal ideation, 31% reported ever having a suicide plan, and 25% reported having made at least one suicide attempt. Significant differences in suicidal ideation were found among the diagnostic groups ($\chi^2=39.2$, $df=9$, $p<0.001$). Specifically, the rates of suicidal ideation in patients with major depression ($\chi^2=21.9$, $df=1$, $p<0.001$), bipolar disorder ($\chi^2=14.7$,

df=1, $p<0.001$), dysthymia ($\chi^2=13.8$, df=1, $p<0.001$), adjustment disorder ($\chi^2=9.5$, df=1, $p<0.005$) and alcohol/substance abuse ($\chi^2=12.8$, df=1, $p<0.001$), although similar to each other (52%–64%), were all significantly greater than the rate in patients with generalized anxiety disorder (18%). Similarly, the rates in patients with major depression ($\chi^2=12.3$, df=1, $p<0.001$), bipolar disorder ($\chi^2=8.9$, df=1, $p<0.005$), and dysthymia ($\chi^2=8.3$, df=1, $p<0.005$) were significantly greater than the rate in patients with organic mental disorder. The rate of suicidal ideation in patients with major depression was significantly greater than the rate in patients with panic disorder ($\chi^2=8.2$, df=1, $p<0.005$). Odds ratios for patients with major depression, bipolar disorder, dysthymia, adjustment disorder, and alcohol/substance abuse having suicidal ideation were, respectively, 8.19, 7.92, 7.76, 5.04, and 6.79 time the odds ratio for patients with generalized anxiety disorder. The odds ratios for patients with major depression, bipolar disorder, and dysthymia were, respectively, 6.2, 6.0, and 5.9 time the odds ratio for patients with organic mental disorder. The odds ratio for patients with major depression was 2.5 time the odds ratio for patients with panic disorder.

Among the 346 patients who reported ever having suicidal ideation, 145 (42%) reported ideation within the past week; there were no significant differences across diagnostic categories ($\chi^2=8.1$, df=9, n.s.). The mean age at first ideation was 25.4 years (SD=13.5). No significant differences were found across diagnostic categories ($F=1.1$, df=9, 260, n.s.) for the age at first ideation. Among the 346 patients reporting current or past ideation, 135 (39%) reported persistent ideation, defined as ideation lasting for at least 7 days. A significant difference was found across diagnostic categories ($\chi^2=28.3$, df=9, $p<0.005$). Specifically, the rates in patients with major depression ($\chi^2=10.8$, df=1, $p<0.005$), schizophrenia ($\chi^2=13.3$, df=1, $p<0.001$), and alcohol/substance abuse ($\chi^2=8.6$, df=1, $p<0.005$) were significantly greater than the rate in patients with adjustment disorder. The rate in patients with schizophrenia was greater than that in patients with dysthymia ($\chi^2=8.9$, df=1, $p<0.005$). Odds ratios for patients with major depression, schizophrenia, and alcohol/substance abuse having suicidal ideation that persisted were, respectively, 4.9, 8.0, and 5.4 time the odds ratio for patients with adjustment disorder. The odds ratio for the patients with schizophrenia having persistent ideation was 5.8 time the odds ratio for patients with dysthymia.

Regarding suicide attempts, significant differences were found among the diagnostic groups ($\chi^2=36.3$, df=9, $p<0.001$). Specifically, patients with major depression differed significantly from those with panic disorder ($\chi^2=12.8$, df=1, $p<0.001$) and those with adjustment disorder ($\chi^2=15.3$, df=1, $p<0.001$). The odds ratio for patients with major depression having attempted suicide was 5.75 time the odds ratio for patients with panic disorder and 4.67 time the odds ratio for patients with adjustment disorder. There were no

significant differences among groups regarding suicide plans ($\chi^2=12.7$, df=9, n.s.).

Subsequent data analyses were done on the 164 patients who reported at least one suicide attempt. The mean age at first ideation of these patients was 22.8 years (SD=12.5). Their mean age at first attempt was 26.2 years (SD=14.3). Seventy-four (45%) reported having made more than one attempt. (Data were missing for some of the items; the percentages were calculated on the number of patients for whom data were available.) The mean number of attempts per attempter was 1.8 and ranged from 1 to 10 attempts. On analyzing just the first reported attempt, we found that 32 (22%) of 148 patients communicated their intent to commit suicide before their attempt and 82 (56%) of 147 communicated their suicide attempt after the attempt. Regarding intentionality of first attempt, 107 (72%) of 148 patients wanted to die and 96 (68%) of 141 expected to die. No significant differences across diagnostic groups were found for the variables of age at first ideation ($F=0.9$, df=9, 260, n.s.), age at first attempt ($F=2.3$, df=9, 139, n.s.), single versus multiple attempts ($\chi^2=11.3$, df=9, n.s.), mean number of attempts ($F=0.9$, df=9, 607, n.s.), communication of intent to commit suicide before attempt ($\chi^2=15.9$, df=9, n.s.), communication of suicide attempt after the attempt ($\chi^2=11.9$, df=9, n.s.), the desire to die ($\chi^2=18.3$, df=9, n.s.), and the expectation to die ($\chi^2=8.2$, df=9, n.s.).

Regarding method of first attempt, 79 (53%) of 150 patients reported overdosing, 25 (17%) reported jumping, 25 (17%) reported wrist cutting, one (1%) reported drowning, five (3%) reported hanging, one (1%) reported shooting, and three (2%) reported using carbon monoxide poisoning (gas). As indicated in table 2, overdosing was the most often used method in all of the diagnostic categories.

DISCUSSION

The findings of this study suggest that suicidal behaviors are widely prevalent among patients seen in a general psychiatric outpatient department; 55% of the sample had a history of suicidal ideation, 39% had persistent suicidal ideation for a minimum of 7 days, and 31% had a clear plan. Not only was there a high prevalence of a past history of suicidal ideation but as many as 43% of these patients had suicidal ideation in the week before answering the survey. Surprisingly, 25% had made at least one previous suicide attempt, and approximately half of the attempters had made multiple attempts; these patients may be particularly vulnerable for completed suicide. Follow-up studies of suicide attempters (8) indicate that 1%–2% complete suicide within a year after an initial attempt, and 1% each year afterward. Furthermore, 40% of patients who commit suicide have made previous suicide attempts (9). The high prevalence of suicide attempts found in our study stands in contrast to the findings of previous outpatient department studies by Linehan and Laffaw (4) and Beck

TABLE 2. Method of First Suicide Attempt Reported by 150 Patients in a Psychiatric Outpatient Department

Diagnosis	Total N	Overdose	Jumping	Wrist Cutting	Drowning	Hanging	Shooting	Carbon Monoxide Poisoning (Gas)	Other	Multiple
Overall	150	79	25	25	1	5	1	3	6	5
Major depression	75	35	17	13	1	1	0	2	3	3
Bipolar disorder	14	7	1	1	0	2	1	1	0	1
Dysthymia	14	11	0	0	0	0	0	0	2	1
Panic disorder	3	2	0	1	0	0	0	0	0	0
Generalized anxiety disorder	5	3	1	1	0	0	0	0	0	0
Schizophrenia	17	9	3	4	0	1	0	0	0	0
Psychotic disorder not otherwise specified	5	3	1	1	0	0	0	0	0	0
Adjustment disorder	7	4	0	2	0	1	0	0	0	0
Organic mental disorder	1	1	0	0	0	0	0	0	0	0
Alcohol or substance abuse or dependence	9	4	2	2	0	0	0	0	1	0

et al. (5), which found prevalence rates of 4% and 10%, respectively. The present study, which represents a large general outpatient department and 651 patients, was nonselective; we evaluated any patient seeking treatment. Thus, the higher rates of suicidal behavior seen in our study may relate to the diverse diagnostic categories represented by the patients studied; many of these patients might actually have been in crisis at the time of evaluation. In contrast, Beck et al. (5) predominately studied patients with mood and anxiety disorders who were referred to a clinic specializing in cognitive therapy. Linehan and Laffaw (4) did not describe their clinic or their patients in detail.

Although suicidal behavior was widely prevalent among all diagnoses assessed in our study, we found that certain diagnostic categories had significantly higher prevalence rates. Most noteworthy was that patients with mood disorders (major depression, bipolar disorder, and dysthymia), adjustment disorder, and alcohol/substance abuse, who had similarly high rates of suicidal ideation (52%–64%), all had significantly higher rates of suicidal ideation than patients with generalized anxiety disorder (18%). A history of suicide attempts was more prevalent in patients with major depression (35%) than those with panic disorder (9%). One limitation of the present study is that we cannot be sure that the suicide attempt occurred simultaneously with the presenting psychiatric disorder. For example, some of the patients with panic disorder may have made attempts before the onset of their panic disorder.

Our finding that patients with anxiety disorders have lower prevalence rates of suicidal behaviors agrees with the finding of Beck et al. (5) that patients with panic disorder had significantly lower rates of suicide attempts and lower suicidal ideation scores than a comparison group of patients with mood disorders. We also found that the rate of suicide attempts in patients with mood disorders was higher than the rate in patients with panic disorder. This is in contrast to the finding of Weissman et al. (10) that the rate of suicide attempts was equivalent in patients with mood and panic disorders. However, the rate of suicide attempters in our

study was substantially higher than that found by Beck et al. (5) for both patients with mood disorders and patients with panic disorders.

The rate of suicide attempts in our patients with panic disorder was lower than that reported by Weissman et al. for patients with panic disorder and higher than that reported by Weissman et al. for patients with major depression (10). It must be underlined that the study by Weissman et al. was part of a large epidemiologic catchment area study that assessed a community sample for a history of lifetime psychiatric diagnoses. Beck et al. (5) have raised a number of issues that may explain the discrepancy between our findings and those of Weissman et al. (10), such as whether findings from a community sample are generalizable to a patient population and whether diagnoses made on the basis of a diagnostic interview schedule administered by lay persons (as was done in the the epidemiologic catchment area study) are as reliable as diagnoses made by mental health professionals who conducted a clinical interview.

Suicide attempts are predominately a secretive event: only 22% of the patients in our study communicated their intent to commit suicide before making an attempt and only 56% communicated the fact that they had made an attempt after doing so. In another study (11) our group found that approximately two-thirds of adolescent suicide attempters in a high school system did not inform anyone before their attempt and that approximately one-third did not tell anyone after their attempt. Since patients are noncommunicative regarding suicide attempts, clinicians must inquire directly about suicidal behaviors. This study suggests that a self-rating survey of suicide behaviors might be particularly useful to address this area. We found that patients who had previously failed to communicate their attempts provided this information in response to survey questions.

It is interesting to note that 41% of our sample identified themselves as Hispanic. Although there was no formal identification of geographic family origin in this study, other research that identified the specific origin of Hispanic patients seeking treatment at our hospital

(12) indicated that the overwhelming majority (91%) are of Puerto Rican descent. The research literature indicates that Puerto Ricans have a higher rate of suicidal behaviors than blacks and whites (13, 14), and this is consistent with our findings. It would be important for future research to determine the precise nature of an association between being of Puerto Rican descent and suicidal behaviors.

A precise clarification of suicidal behaviors in general psychiatric outpatients has important clinical implications. With the continuing emphasis on deinstitutionalization, outpatient departments are providing services for patients who in the past would have been hospitalized. These facilities are in many cases treating more acutely ill patients, a fact that may well be reflected in the high rate of suicidal behaviors in our study group. Knowledge of diagnoses that are associated with suicidal ideation, plans, and attempts is crucial for the clinician who must decide on treatment, including medication and possible hospitalization. Knowledge of the prevalence of suicidal behaviors in a general outpatient department would have a direct impact on decisions regarding how to use staff resources in such a facility.

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Dawn Simulation Treatment of Winter Depression: A Controlled Study

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Objective: This study sought to determine whether dawn simulation was superior to a shorter dimmer "placebo" dawn signal in treating winter depression. **Method:** In a randomized, parallel design, 22 patients with winter depression were treated with either 1 week of a 2-hour dawn simulation peaking at 250 lux or 1 week of a 30-minute dawn simulation peaking at 0.2 lux. The subjects were told that they would receive either a "gradual" dawn or a "rapid" dawn reaching an intensity that would be dimmer than standard bright light treatment. At the end of both the baseline week and the treatment week, subjects were assessed in a blind manner with the Hamilton Rating Scale for Depression. Analysis of covariance was used to compare the two dawn treatments. **Results:** The 2-hour, 250-lux dawn simulation resulted in Hamilton depression scale scores that were significantly lower than scores after the 30-minute, 0.2-lux dawn simulation. **Conclusions:** This study indicates that dawn simulation is an effective treatment for winter depression.

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Bright light (greater than 2500 lux) has been shown to be more effective than dim light (less than 500 lux) control conditions in the treatment of winter depression (1-5). However, Terman et al. (6-8) have reported preliminary data concerning dawn simulation, a dim light that gradually increases in illuminance to a peak of only 100-500 lux before subjects awaken. In an uncontrolled study of dawn simulation, they noted clinical improvement in six of eight subjects.

Using a parallel design, the present study compared a gradual 2-hour dawn peaking at 250 lux with a 30-minute dawn peaking at 0.2 lux, a placebo condition.

METHOD

Subjects were recruited through advertisements and through publicity concerning our program. They fulfilled

criteria for major depressive episode according to DSM-III-R, as well as primary affective disorder according to Feighner criteria (9). They also fulfilled Rosenthal criteria for seasonal affective disorder (1). Subjects reported regularly occurring fall-winter depressions (with at least two occurring during consecutive winters) that remitted during the spring or summer. In addition, no psychosocial variables could account for the regular changes in mood. All had hypersomnia as part of their winter depression. Hypersomnia was defined as sleeping at least an hour more during the winter depression than during the euthymic summer period. All subjects were free of any psychotropic medication for at least 2 weeks before the study. Subjects gave written informed consent.

During the 2-week outpatient study, subjects were asked to sleep only between the hours of 9:00 p.m. and 6:00 a.m. and to keep a log of their sleep. The first week was a baseline week during which no light treatment was administered. At the beginning of the second week the subjects were randomly assigned to one of two dawn simulation conditions, either a gradual (approximately $2.2 \log_{10}$ lux/hour) dawn simulation over a 2-hour period (4:00 a.m. to 6:00 a.m.) that peaked at 250 lux (similar to room light level) or a gradual (approximately $2.6 \log_{10}$ lux/hour) increase in light intensity over a 30-minute period (5:30 a.m. to 6:00 a.m.) that peaked at an illuminance of 0.2 lux (similar to moonlight). The 30-minute dawn gave the same signal as the first 30 minutes of the 2-hour dawn. The 2-hour dawn

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approximates the dawns of April 21 and August 19 in Seattle (47° latitude) (Daylight Savings Time). During both weeks the subjects slept at home. The randomization was stratified according to sex and quarter of the menstrual cycle. Subjects were told that they would be randomly assigned to receive either a gradual 2-hour dawn or a rapid 30-minute dawn and that the final illuminance of both would be much dimmer than standard bright light treatment.

Both dawn light treatments used a 75-watt incandescent reflector flood that was four feet from the pillow. The intensity of the light at this distance was measured with a photometer. Subjects were asked to sleep until 6:00 a.m., at which time they were awakened by an alarm. If they awoke before 6:00 a.m., they were asked to avoid looking at the lights, close their eyes, and try to go back to sleep. The incandescent light was plugged into a dawn simulator device that created a gradually increasing voltage to the incandescent light starting at a time that could be specified. Subjects used these lights daily during the treatment week.

The subjects' bedrooms were required to be dark. If a street light or security light did shine through the bedroom windows, subjects were given a sheet of black plastic with which to cover the windows. They were asked to turn off any night lights or hall lights that might shed light into the bedroom. These patients with hypersomnia typically slept throughout the night and did not need to get up to use the bathroom. If they did get up to use the bathroom, they attempted to do so with the aid of only a night light in the bathroom. During the study they were advised to avoid morning sunlight before 8:00 a.m.

Expectations of the response to gradual 2-hour dawn simulation and rapid 30-minute dawn simulation were assessed at baseline. Subjects rated their expected response to rapid dawn simulation and to gradual dawn simulation on a global scale (1=worse, 2=no change, 3=slight improvement, 4=much improvement, 5=very much improvement). At the end of each light treatment subjects rated their response to light treatment on the same global scale.

Subjects were rated in a blind manner by experienced psychiatrists using the Structured Interview Guide for the Hamilton Depression Rating Scale, Seasonal Affective Disorder Version (10)—which includes the 21-item Hamilton Rating Scale for Depression (11) and adds eight supplementary questions (seasonal affective disorder subscale) concerning atypical depressive symptoms such as hypersomnia and increased appetite. The ratings took place at the end of each week of the study.

Subjects systematically were asked about possible side effects such as early morning awakening, headache, agitation, evening drowsiness, irritability, and tight muscles. Each item was rated as absent, mild, moderate, or severe.

Statistical analyses included analysis of covariance (ANCOVA), two-way, repeated-measures analysis of variance (ANOVA), Fisher's exact test, and unpaired *t* tests.

RESULTS

Of 27 subjects, 14 (10 women and four men) were randomly assigned to the 2-hour, 250-lux dawn; 13 (nine women and four men) were randomly assigned to the 30-minute, 0.2-lux dawn. The two dawn conditions had similar numbers of women in the first, second, third, and fourth quarters of the menstrual cycle (2-hour dawn: *N*=2, 1, 2, 3; 30-minute dawn: *N*=3, 1, 2, 1) and similar numbers of women without menses (2-hour dawn: *N*=2, 30-minute dawn: *N*=2). Four of the subjects had a history of hypomania (one in the 2-hour dawn group and three in the 30-minute dawn group). Fourteen of the 27 subjects had taken either antidepressant or antianxiety medication for previous depressions (six in the 2-hour dawn group and eight in the 30-minute dawn group). One in each group had a history of alcohol abuse; one, a man, had been abstinent for 5 months, and the other, a woman, had been abstinent for 1 month. The subjects were studied between Nov. 20, 1990, and March 27, 1991.

One subject from the 2-hour dawn group failed to return for the last visit. Four subjects from the 30-minute dawn group dropped out of the study during the treatment week. One woman stopped the dawn treatments after 4 days because of lack of response. The woman with a history of alcohol abuse began drinking again. Erratic electrical power to the home of one woman prevented the dawn simulation on two mornings. One man failed to return after the treatment week because of a busy schedule. These five dropouts did not differ from those who completed the study in age, sex, or baseline depression ratings. All subsequent analyses are based on the 22 subjects who completed the protocol.

For the 13 subjects who completed the 2-hour dawn simulation, the mean age was 35.0 years (*SD*=8.8); for the nine subjects who completed the 30-minute dawn simulation, the mean age was 35.7 years (*SD*=11.3). At baseline the overall expectations of those who completed the study for the 2-hour and the 30-minute dawn simulations were similar (mean=3.27, *SD*=0.77, versus mean=3.18, *SD*=0.59; *t*=0.8, *df*=21, *n.s.*). Baseline expectations for the treatment actually received were not statistically different (2-hour dawn: mean=3.46, *SD*=0.88; 30-minute dawn: mean=3.11, *SD*=0.33; *t*=1.14, *df*=20, *n.s.*).

The global improvement rating after treatment was significantly better for the 2-hour dawn simulation than for the 30-minute dawn simulation, using ANCOVA with baseline expectations for the treatment received as the covariate (mean=4.31, *SD*=0.48, versus mean=3.00, *SD*=0.50; *F*=21.1, *df*=1, 19, *p*<0.001).

The 2-hour dawn group and the 30-minute dawn group did not have significantly different baseline scores for the Hamilton depression scale (*t*=0.60, *df*=20, *n.s.*) and the seasonal affective disorder subscale (*t* with unequal variances=1.36, *df*=10.83, *n.s.*). Depression rating scores after treatment with 2-hour dawn simulation were significantly lower than scores after

30-minute dawn simulation, using ANCOVA with the baseline scores as the covariate (table 1). Both the Hamilton depression scale and the seasonal affective disorder subscale ratings were significantly lower than baseline scores in the 2-hour dawn group; in the 30-minute dawn group depression ratings were lower than baseline but to a nonsignificant degree.

Of the subjects who received the 2-hour dawn simulation, one had a slight headache and five experienced mild early morning awakening, usually a brief awakening during the first few days of treatment between 5:00 a.m. and 6:00 a.m. However, one woman experienced significant hypomania, with early morning awakening, sleeping only 4 hours per night, racing thoughts, improved mood, and increased energy. She experienced uncomfortable agitation for 1 day. This subject had no personal or family history of mania or hypomania. In contrast, only one subject who received the 30-minute dawn simulation experienced mild early morning awakening. The proportion of subjects who experienced insomnia was not significantly greater in the 2-hour dawn group (six of 13 subjects) than in the 30-minute dawn group (one of nine subjects) (Fisher's exact test, n.s.). Among those who received 2-hour dawn simulation, the Hamilton depression scale and seasonal affective disorder subscale scores did not differ significantly (Hamilton depression scale: $t=0.47$, $df=11$, n.s.; seasonal affective disorder subscale: $t=1.20$, $df=11$, n.s.) in those who had early morning awakening ($N=6$, mean=4.8, $SD=3.2$, and mean=5.6, $SD=4.5$) and in those who did not ($N=7$, mean=6.0, $SD=5.5$, and mean=2.8, $SD=3.7$).

Complete sleep logs were available for 11 of the 13 subjects who received 2-hour dawn simulation and all nine who received 30-minute dawn simulation. The sleep logs revealed that the two groups were able to comply with the sleep schedules. A two-way, repeated-measures ANOVA revealed no differences in sleep duration during the baseline and treatment weeks ($F=0.21$, $df=1, 18$, n.s.), no differences between the two treatment groups ($F=0.17$, $df=1, 18$, n.s.), and no interaction between order and group ($F=2.2$, $df=1, 18$, n.s.).

DISCUSSION

The response to the 2-hour, 250-lux dawn was superior to the response to the 30-minute, 0.2-lux placebo dawn in patients with winter depression and is similar to responses observed with bright light boxes. The drop in the Hamilton depression scale scores from 17.1 to 5.5 is similar to the drop from 17.8 to 8.1 seen in pooled ($N=172$) data from studies using morning bright light therapy (5). Morning bright light appears to be superior to evening bright light (5, 12), particularly for subjects with winter depression and hypersomnia (13). The response to the 2-hour, 250-lux dawn is also similar to responses observed in earlier studies of dawn simulation. In an open design, Terman et al. (7) found that dawn simulation lowered Hamilton depression scale

TABLE 1. 21-Item Hamilton Rating Scale for Depression and Seasonal Affective Disorder Subscale Scores for Subjects With Winter Depression Treated With a 2-Hour, 250-Lux Dawn Simulation or a 30-Minute, 0.2-Lux Dawn Simulation

Item	2-Hour, 250-Lux Dawn (N=13)		30-Minute, 0.2-Lux Dawn (N=9)	
	Mean	SD	Mean	SD
Hamilton depression scale				
Baseline	17.1	4.6	18.6	7.0
Treatment	5.5 (5.6 ^a) ^{b,c}	4.5	11.1 (10.9 ^a) ^d	4.9
Seasonal affective disorder subscale				
Baseline	13.1	3.1	16.1	6.2
Treatment	4.3 (4.6 ^a) ^{e,f}	4.2	8.8 (8.3 ^a) ^g	3.5

^aCorrected mean.

^b $F=7.0$, $df=1, 19$, $p<0.05$ (ANCOVA).

^c $t=7.0$, $df=12$, $p<0.01$ with Bonferroni correction (two-tailed test).

^d $t=3.2$, $df=8$, $p=0.08$ with Bonferroni correction (two-tailed test).

^e $F=4.38$, $df=1, 19$, $p=0.05$ (ANCOVA).

^f $t=10.6$, $df=12$, $p<0.01$ with Bonferroni correction (two-tailed test).

^g $t=3.0$, $df=8$, $p<0.10$ with Bonferroni correction (two-tailed test).

scores from 13.1 to 4.9 in eight subjects with winter depression. Avery et al. (14) found that in 13 subjects with winter depression, a 2.5-hour dawn peaking at 275 lux lowered Hamilton depression scale scores from 17.7 to 5.9 and a 10-minute dawn peaking at 275 lux lowered Hamilton depression scores from 17.2 to 7.0. In an open study Jacobsen (15) treated 25 patients with major affective disorder and with complaints of either oversleeping or difficulty getting up without prewaking light. A 150-watt incandescent floodlight came on 10 minutes before the preselected wake-up time. Sixty percent reported a full response and 36% reported partial response. However, the presence of concomitant medications and absence of a control condition cloud interpretation of the Jacobsen study.

Because of significant placebo responses often observed among depressed subjects (16), the possibility that the therapeutic effect of the 2-hour dawn simulation can be accounted for by a placebo effect should be explored. A true placebo condition for dawn simulation, as for other light therapies (16), may be difficult to define, since subjects are able to see the treatment condition upon awakening. The baseline expectations for the two dawn simulations were similar. However, because we did not want to bias the subjects against the dim dawn by showing both dawns, we may not have been measuring true expectations. Because of the parallel design, both groups were blind to final intensity of the dawn simulation received by the other group.

However, the shorter, dimmer dawn acted like a convincing control condition. The drop in Hamilton depression scale scores from 18.6 to 11.1 in the control condition is better than the drop in Hamilton depression scores of 23.4 to 20.0 seen in pooled ($N=77$) data from studies using a dim light box control and is similar to the decrease (18.0 to 10.1) seen in pooled ($N=143$) data from studies of 2 hours of evening bright light (5).

Although patients with recurrent depression may have high placebo response rates (17), patients with winter depression do not respond particularly well to placebo medication. O'Rourke et al. (18) found that Hamilton depression scale scores dropped from 21 to 17 in 18 patients with seasonal affective disorder in response to a placebo pill. McGrath et al. (19) found that Hamilton depression scale (17-item) scores dropped from 15.5 to 14.2 after 1 week of a placebo regimen. Rosenthal et al. (20) found that Hamilton depression scale scores dropped from 23.1 to 18.0 after 1 week of a placebo regimen. The dim short dawn may have been effective; but, without a less effective control condition, its efficacy remains unknown. Because of the superiority of the 2-hour dawn to the short dim dawn, the response to the 2-hour, 250-lux dawn was probably not simply a placebo effect.

Dawn simulation is an innovative technique developed by the pioneering research of Terman et al. (6–8); it differs from standard phototherapy in that dawn simulation occurs earlier, while the subject is asleep, and with a gradually increasing dim illuminance. The dawn simulation used in the present study differed from that described by Terman et al. They used a computer that employed a photosensor feedback loop and light-attenuation mechanism that approximated a dawn signal according to a sophisticated algorithm. The present study used a dawn simulator that created a gradual illumination ramp which may not be as precise an approximation of a natural dawn as the Terman dawn simulator. Whether a precise replication of a natural dawn is necessary is not clear; in nature the dawn signal reaching the eyes may be influenced by changing clouds, trees, sleeping position, and so forth. Further, the duration of the dawn varies with latitude (8). The relative importance of the peak illuminance and the shape of the dawn signal is not known. In nine subjects, the response to a gradual 2.5-hour dawn peaking at 275 lux was similar to the response to a 10-minute dawn peaking at 275 lux in average depression ratings; however, some subjects clearly preferred the 2.5-hour dawn, while others preferred the 10-minute dawn (14). Terman et al. (7) have hypothesized that the shape of the dawn signal may be important for therapeutic effect; they found that a bright light automatically switched on an hour before the usual wake-up time was clearly not clinically effective for three subjects with winter depression. Further research needs to clarify which treatment variables are important and to address the heterogeneity of responses to dawn signals.

Since the bright light therapy seems to work through the eyes (21), how can the efficacy of dawn simulation be explained in subjects whose eyes are closed? The eyelids are translucent to light transmitting about 10% in the red end of the visible spectrum (above 700 nm), with the transmittance declining to 1%–2% in the green and blue end (below 600 nm) (22). In the visible spectrum, the spectral distribution of the incandescent lights used in the present study shows a predominance of red over blue and green. However, dim light acti-

vates the rods that respond primarily to green light (23).

Since dim light boxes using intensities of 100–400 lux have been found relatively ineffective (24), how can one explain the efficacy of a dawn signal with a maximum intensity of 250 lux which is partially blocked by the eyelids? Retinal sensitivity is particularly great during the early morning hours (25, 26). It has been hypothesized that the phase-shifting effect of bright light therapy is its mechanism of action (27–30); dawn simulation may work in a similar way (7). Low levels of light have been shown to shift circadian rhythms (31–33). Terman et al. (7) found that in two patients, the nocturnal melatonin rhythm was phase-advanced (shifted earlier) by dawn simulation up to 250 lux relative to dim (<1 lux) baseline conditions. However, other mechanisms of action, unrelated to phase-shifting, cannot be excluded and should be explored.

Dawn simulation is not without side effects. One of the 13 subjects who received the 2-hour dawn simulation became hypomanic; in a previous study (14) hypomania was seen in one of 12 subjects receiving a 2.5-hour, 275-lux dawn. Otherwise, the dawn simulation in the present study was well tolerated except for some brief awakenings from 5:00 a.m. to 6:00 a.m. in five subjects. In contrast, Avery et al. (34) found that a 2-hour dawn peaking at 1700 lux resulted in at least mild early morning awakening in all seven subjects and severe early morning awakening in three. Thus, an "overdose" of dawn is possible. Whether this effect is secondary to an extreme phase-advance of the circadian clock is not known.

The sleep interruption that can be seen with dawn simulation raises the possibility that dawn simulation might work through partial sleep deprivation, an effective treatment for depression (35). However, the improvement seen among those who reported early morning awakening with the 2-hour dawn was similar to that seen among those without early morning awakening. The sleep logs showed that sleep duration was similar in those who received the 30-minute dawn simulation and those who received the 2-hour dawn simulation. In addition, our previous studies show that those who experienced severe early morning awakening from the dawn simulation did not do as well as those who were not awakened early by the dawn signal (14, 34). These data argue against a role of sleep deprivation in the effectiveness of dawn simulation.

Further controlled studies of the efficacy, safety, and mechanism of action of dawn simulation are necessary.

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A Double-Blind Crossover Trial of Imipramine and Phenelzine for Outpatients With Treatment-Refractory Depression

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***Objective:** Data from controlled studies concerning the response rates of patients to a second antidepressant medication after they have been unresponsive to a systematic trial of another antidepressant are extremely useful to clinicians for rational prescription of pharmacotherapy. Such information allows making an accurate prognosis, sustaining realistic hope in the patient, and achieving the best possible therapeutic outcome. This study was designed to add to the scanty literature available on this subject. **Method:** Eighty-nine mood-reactive, nonmelancholic, mainly chronically depressed outpatients at a university research clinic who were unresponsive to vigorous double-blind trials of imipramine or phenelzine were crossed over to treatment with the other drug under double-blind conditions. **Results:** Of 46 patients previously unresponsive to imipramine who completed phenelzine treatment, 31 (67%) responded to phenelzine. Of 22 patients previously unresponsive to phenelzine who completed imipramine treatment, nine (41%) responded to imipramine. The difference in response rates was statistically significant. Even after they had shown no response to 7 weeks of placebo and 6 weeks of imipramine treatment, 10 (83%) of 12 patients who then completed treatment with phenelzine responded. **Conclusions:** These data suggest that among chronically ill, mood-reactive depressed patients with many symptoms of atypical depression, phenelzine is strikingly effective in those who have been nonresponders to imipramine and should be tried in such patients.*

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While clinical reports concerning treatment of patients unresponsive to an initial drug trial abound (1-11), data from controlled studies are scarce. An accurate estimate of the probable effectiveness of various treatment strategies, ideally based on controlled study data, is crucial to effective treatment. It assists the clinician in developing a rational plan of treatment, giving an accurate prognosis, and sustaining the patient's hope.

A number of reports of uncontrolled studies concerning patients unresponsive to tricyclic treatment

have indicated a good response to monamine oxidase inhibitor (MAOI) antidepressants. Schatzberg et al. (11) reported on a series of patients unresponsive to treatment who were referred to a specialized depression treatment unit. Of 57 patients they treated with an adequate course of a tricyclic antidepressant, 15 (26%) showed little or no improvement and were then given open treatment with an MAOI. Nine (60%) of the 15 responded favorably to the MAOI. Although clinical lore has suggested that patients with endogenous depression or melancholia may not respond well to MAOIs, seven (78%) of nine patients in that study who were considered to have definite endogenous depression responded to MAOI treatment.

Nolen et al. (12) treated nonresponders to cyclic antidepressants with either oxaprotiline or fluvoxamine, nontricyclic selective reuptake inhibitors of noradrenaline and serotonin, respectively. Of patients who were unresponsive to one of these, 27% (14 of 52) responded when crossed over to the other drug. In a second study (13), 33 nonresponders from the first study were treated with L-5-hydroxytryptophan, nomifensine, or

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the MAOI tranylcypromine. There was a 50% response rate to the MAOI but only a 5% response rate to the other treatments.

In a retrospective study of nondelusional unipolar inpatients unresponsive to well-documented trials of imipramine, Roose et al. (14) found that nine of 10 responded to subsequent ECT, and all five who received MAOIs had a "robust response."

In the only controlled study of which we are aware, Thase et al. (15) reported that nine of 12 patients with anergic bipolar depression who were unresponsive to imipramine responded when crossed over to tranylcypromine treatment. Only one of four nonresponders to tranylcypromine responded when crossed over to imipramine in that study.

While suggesting that MAOIs are effective in both melancholic and nonmelancholic depressions refractory to tricyclic treatment, most of these studies were not double-blind and therefore not definitive. To our knowledge, the study reported by Thase et al. is the only one concerning response to tricyclic antidepressants of patients unresponsive to MAOIs.

The present report grew out of studies of patients with nonmelancholic depression, most of whom met Columbia criteria for atypical depression. These criteria require mood reactivity (significant mood response to favorable events) as well as two of the following four associated symptoms: overeating, oversleeping, severe loss of energy, and pathologic sensitivity to interpersonal rejection (16). When it became clear that not all mood-reactive, nonendogenous patients met these criteria, our studies were expanded to include all mood-reactive patients who were nonmelancholic according to DSM-III criteria (17).

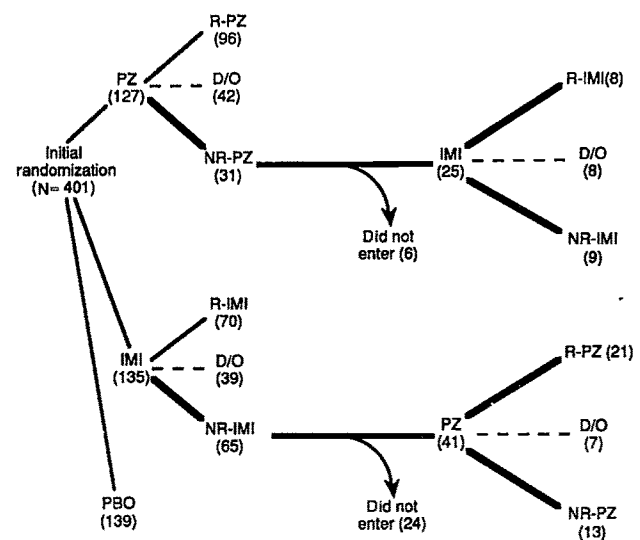
The questions addressed by this report are the following.

1. Among patients not responsive to an adequate trial of either imipramine or phenelzine, what was the response rate to the alternate active drug?
2. Did patients considered to be nonresponders have an adequate trial of treatment as measured by dose and plasma levels or level of MAO inhibition? Was the level of MAO inhibition associated with response?

METHOD

The subjects included in this report were nonmelancholic outpatients with mood-reactive depression who participated in a controlled clinical trial comparing phenelzine, imipramine, and placebo. The primary outcome data for 329 patients on the first treatment received have been reported in a series of articles (18–20). This report presents previously unreported data for the subset of 89 patients (27%) who were nonresponders to the first active drug they received and who completed a trial of the other active drug under double-blind conditions. A partial preliminary report of these data has been made (21). Patients who failed to improve when taking either drug after an initial lack of response to a

FIGURE 1. Study Design and Patient Flow for Nonresponders to the First Active Medication in a Crossover Trial of Imipramine and Phenelzine for Depressed Outpatients^a



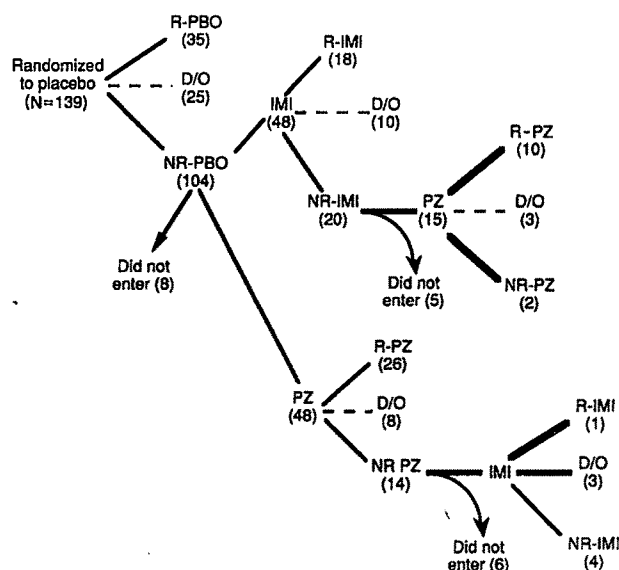
^aPZ=phenelzine, IMI=imipramine, PBO=placebo, D/O=dropouts, R=responders, NR=nonresponders. The response rate for the 17 completers of the imipramine trial was 47%; the response rate for the 34 completers of the phenelzine trial was 62%.

placebo trial were also included so as to have the largest possible sample of nonresponders to each active drug.

The study design, inclusion and exclusion criteria, dosage schedules, and description of the study group have been published previously (18–20). Briefly, all patients met the Research Diagnostic Criteria (22) for major, minor, or intermittent depression or the DSM-III criteria for depressive disorders (major depression, dysthymia, or atypical depression), were mood reactive, were between the ages of 18 and 65 years, and gave written informed consent.

The Clinical Global Impression (CGI) (23) improvement score was used throughout the study to rate patients' responses. Patients rated "much improved" or "very much improved" after 6 weeks were considered responders. This criterion was applied conservatively, since patients received this rating only if there was marked improvement. The 21-item Hamilton Rating Scale for Depression (24) and the 90-item Hopkins Symptom Checklist (SCL-90) (25) were used to assess severity of depression. Chronicity of depression was rated on a 4-point scale (1=mostly well, 2=depressed about half the time, 3=depressed most of the time, and 4=always or almost always depressed).

The design and patient flow of the study are presented in figure 1. After a 10-day single-blind placebo washout, unimproved patients were randomly assigned to 6 weeks of treatment with phenelzine, imipramine, or placebo (phase 1). Nonresponders to either of the active drugs were crossed over under blind conditions to the other treatment after 5 days of drug tapering and a 9-day washout period. Double-blind



Continuous data were analyzed by analysis of covariance (ANCOVA), with baseline score after washout at the end of the first active treatment as the covariate.

Variable	N	%
White race	81	91
Female sex	47	53
Married	26	29
Social class I-III	67	75
Finished college	37	42
Diagnosis ^a		
Major depression	64	72
Intermittent depressive disorder	41	46
Bipolar II disorder	12	13
Definite atypical depression	45	51
Probable atypical depression	23	26
Ever hospitalized	13	15
Ever attempted suicide	21	24
Panic present	47	53

RESULTS

Figure 1 summarizes the outcome in phase 2 for all patients who were unresponsive to an adequate trial of either active drug in phase 1. Of all 41 patients unresponsive to imipramine who crossed over to phenelzine (intention-to-treat analysis), 21 (51%) responded. Of the 34 completers (i.e., those who had an adequate

trial), 21 (62%) responded to phenelzine. Of the 25 patients unresponsive to phenelzine who crossed over to imipramine (intention-to-treat analysis), eight (32%) responded. Of the 17 who completed an adequate trial, eight (47%) responded. The response rates to the two drugs were not significantly different in either the intention-to-treat analysis ($\chi^2=1.61$, $df=1$, n.s.) or the completer analysis ($\chi^2=0.49$, $df=1$, n.s.).

Outcome for Placebo Nonresponders Also Unresponsive to the First Active Drug Received

Figure 2 summarizes the data for subjects not responsive to placebo and to the first active drug. Of 48 placebo nonresponders given phenelzine in phase 2, 14 were nonresponders to phenelzine also. Of the eight of these who entered a trial of imipramine in phase 3 (intention-to-treat analysis), one (12.5%) responded; of the five who completed this trial, one (20%) responded. Of 48 placebo nonresponders given imipramine in phase 2, 20 were nonresponders to imipramine also. Of the 15 of these who were given phenelzine in phase 3 (intention-to-treat analysis), 10 (67%) responded; of the 12 who completed this trial, 10 (83%) responded. The difference in response to the two drugs when they were given as the third treatment was significant both by the intention-to-treat analysis ($p<0.05$, Fisher's exact test) and the completer analysis ($p<0.05$, Fisher's exact test). This suggests that the differential efficacy of the two active drugs may be most clearly seen when the comparison is between patients who have previously been unresponsive to a long double-blind trial of placebo. Further, when most placebo responders were removed from the analyses, imipramine was relatively ineffective for previous nonresponders to phenelzine, while phenelzine was effective for previous nonresponders to imipramine, at least in this small study group.

Combined Outcome for All Patients Crossed Over Between Active Drugs

If all patients unresponsive to imipramine who completed a trial of phenelzine in either phase 2 or phase 3 are combined using the intention-to-treat samples, 31 of 56 (55%) responded to phenelzine. If the same is done for patients unresponsive to phenelzine who completed a trial of imipramine in either phase, nine of 33 (27%) responded (overall Mantel-Haenszel $\chi^2=6.44$, $p=0.01$). The between-trial lack of homogeneity was not significant ($\chi^2=1.72$, $p=0.19$).

Since all of these findings were based solely on CGI scores as the measure of response, the other standard rating scale scores were analyzed to validate these findings. First, rating scale scores for nonresponders to each of the active drugs were compared to test whether these patients were equivalently symptomatic before being crossed over to the other treatment. The mean Hamilton depression scale, CGI, and SCL-90 posttreatment scores of the patients who were not responsive to treat-

TABLE 2. Scores After the Second Active Drug Treatment for Nonresponders to the First Active Drug in a Crossover Trial of Imipramine and Phenelzine for Depressed Outpatients

Scale	Score ^a				F (df=1, 65)	p
	Patients Receiving Imipramine (N=22)		Patients Receiving Phenelzine (N=46)			
	Mean	SD	Mean	SD		
Clinical Global Impression Severity	3.1	1.2	2.3	1.0	8.35	0.005
Improvement	2.9	1.2	2.1	1.0	7.11	0.01
Hamilton Rating Scale for Depression (total score)	10.1	7.0	7.7	4.8	2.47	0.12
SCL-90						
Somatization	1.8	0.6	1.8	0.6	0.00	n.s.
Interpersonal	2.2	0.6	1.8	0.7	4.59	0.04
Depression	2.5	0.9	2.1	0.9	2.49	0.12
Anxiety	2.0	0.6	1.8	0.7	2.28	n.s.
Phobia	1.7	0.7	1.5	0.6	2.15	n.s.
Psychoticism	1.7	0.4	1.5	0.4	4.99	0.03
Obsessive- compulsive	2.3	0.7	2.1	0.8	0.54	n.s.
Paranoia	1.8	0.4	1.7	0.8	0.02	n.s.
Hostility	1.7	0.6	1.4	0.6	3.76	0.06
Summary score	17.8	4.0	15.7	4.9	2.64	0.11

^aAll scores are means adjusted for end-of-treatment scores at the end of the first active treatment by analysis of covariance.

ment with imipramine in either phase 2 or phase 3 were compared by ANCOVA to the posttreatment scores of the patients unresponsive to phenelzine in either phase. Patients were compared after treatment, that is, at the point of nonresponse according to their scores on the CGI, the Hamilton depression scale, and the nine SCL-90 subscales and their total SCL-90 scores. Only scores on the somatization subscale of the SCL-90 differed significantly at the $p=0.05$ level: imipramine-treated patients were rated as slightly worse on that subscale. This indicates that nonresponders to both treatments were equivalently symptomatic at the point at which they were considered nonresponders and were to be crossed over to the other drug.

Next, rating scale scores were compared at the end of the second active drug treatment period. Table 2 summarizes the posttreatment differences among all patients who were considered nonresponders to the first active drug they received and then completed treatment with the second active drug. The phenelzine-treated patients were significantly less severely ill after treatment according to the CGI, which supports the clinical judgment on the CGI improvement score. On the Hamilton depression scale and the SCL-90 subscales, out of 11 comparisons, there were two significant differences and four differences approximating trends in which response to phenelzine was superior. On all but one subscale used, phenelzine-treated patients were rated as less ill, although most differences did not reach statistical significance.

Dosage, Blood Level, and MAO Inhibition

The mean daily dose of imipramine for all nonresponders to imipramine, whether they received imipramine first or placebo first, was 274 mg (SD=45). The mean dose did not differ between those who received the active drug first (mean=272 mg/day, SD=46) and those who received placebo first and then the active drug (mean=279 mg/day, SD=42) ($t=0.71$, $df=76$, n.s.). The mean combined plasma level of imipramine plus desipramine for nonresponders to imipramine was 382 ng/ml (SD=29) (N=69). These data cannot be used to test hypotheses about plasma level and response, since the dose was raised for nonresponders. However, the levels suggest that patients who failed to respond to imipramine were vigorously treated.

The mean daily dose of phenelzine for nonresponders to phenelzine was 75 mg (SD=17). The mean platelet MAO inhibition level was 84% (SD=21%) (N=37). The mean MAO inhibition level for responders to treatment (N=27) was 84% and for nonresponders (N=10) 83%. Response rates were compared using a cutoff of 80% inhibition, a level below which poorer phenelzine response has been reported (28). Two (20%) of the 10 nonresponders had levels below 80%, and six (22%) of the 27 responders had such levels (nonsignificant difference, Fisher's exact test). This fails to confirm a relation between level of platelet MAO inhibition and clinical outcome in our study group, which is in agreement with a recent study which found that end-of-treatment platelet MAO inhibition was not predictive of treatment outcome with phenelzine (29).

DISCUSSION

These data indicate that under controlled double-blind conditions, relatively chronically depressed, mood-reactive outpatients with atypical symptoms who are unresponsive to treatment with tricyclic antidepressants show a clinically significant rate of response to MAOIs. We were able to find only one other published study in which patients unresponsive to an MAOI or to a tricyclic received the other class of drug under double-blind conditions (15).

The absence of a sufficient number of patients with nonatypical depression (N=9) makes it impossible to conclude from our data whether this finding can be generalized to nonatypical depression. Clinical reports by Schatzberg et al. (11), Roose et al. (14), and McGrath et al. (30, 31) as well as a controlled study by Janicak et al. (32) strongly suggest that MAOIs are effective for patients with endogenous or melancholic depression, but no data from crossover studies for this group have been published.

A more disappointing response to imipramine was seen among patients unresponsive to an adequate trial of phenelzine, similar to the finding of Thase et al. (15) for bipolar patients unresponsive to tranylcypromine. It is important to note, however, that the response rates

of 27% (counting dropouts as nonresponders) or 41% (for completers) may still be clinically significant in this group of patients, which probably contained very few placebo responders, since no significant improvement had occurred during the preceding 7–13 weeks of treatment. The absence of an adequate control for time or the possible effects of continued treatment with the first drug are a limitation of the data presented here.

We could find no demographic or diagnostic variable that differentiated completers of the crossover trial from those who did not enter or entered and dropped out. While this gives some support to generalizing these findings to the whole group of patients who began the trial, the large degree of attrition means that the findings can be properly applied only to those patients who completed the crossover trial. Also, since there are no systematic data showing that response differs to different drugs of the tricyclic or of the MAOI class, these findings may generalize to other drugs of either class, but further studies would be necessary to establish this. The dosage, blood level, and MAO inhibition data indicate that the large majority of nonresponders to either drug had taken adequate doses and that lack of response was not an artifact of inadequate treatment.

We could find no diagnostic predictors of treatment outcome, as the group sizes were too small for meaningful comparisons of subgroups. In the whole study group, we have shown that the presence of any atypical symptom of depression predicts poorer outcome with imipramine than the outcome for patients with no atypical symptom (33) and that imipramine does not worsen atypical depression merely by its sedative side effect profile (34). Previous work with primary treatment outcome predictors has not validated our preliminary finding that panic attacks were predictive (19).

The clinical relevance of these data is that chronically ill outpatients with atypical depression who are unresponsive to a trial of a tricyclic antidepressant should have a trial of an MAOI. Available data on the prevalence of atypical depressive symptoms suggest that they are common both in inpatient and outpatient settings. The most frequent clinical practice with such patients is to try another heterocyclic antidepressant (fluoxetine, bupropion, etc.). While this may be efficacious, there are few systematic data available to assess the probability of response. Unfortunately, many psychiatrists do not use MAOI antidepressants at all because of the fear of hypertensive crises, but in the light of our data, this may deprive some patients who are unresponsive to tricyclics of a significant benefit.

The usefulness of a trial of tricyclics for nonresponders to MAOIs is less clear. We believe that it is justified even by the modest response we saw in this study, because the placebo response is probably very low in this group, and therefore much of the 27% response rate probably constituted a true pharmacologic response. To establish this, however, drug crossover would have to be compared, in systematic studies, with other strategies such as potentiation with lithium carbonate.

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Association of Depression With 10-Year Poststroke Mortality

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***Objective:** Depression has been linked to higher than expected mortality from natural causes, particularly among elderly patients with physical illness. The authors examined the effect of depression on mortality among a group of stroke patients followed up for 10 years. **Method:** A consecutive series of 103 patients was assessed for major or dysthymic (minor) depression approximately 2 weeks after stroke with the use of a structured mental status examination and DSM-III diagnostic criteria. Vital status was determined for 91 of these patients 10 years later. **Results:** Forty-eight (53%) of the 91 patients had died. Patients with diagnoses of either major or minor depression were 3.4 times more likely to have died during the follow-up period than were nondepressed patients, and this relationship was independent of other measured risk factors such as age, sex, social class, type of stroke, lesion location, and level of social functioning. The mortality rate among depressed patients with few social contacts was especially high: over 90% had died. **Conclusions:** These results indicate that depressed mood following stroke is associated with an increased risk of subsequent mortality. Patients who are depressed and socially isolated seem to be particularly vulnerable.*

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The link between psychiatric disorder and mortality from natural causes (i.e., other than suicide and accidents) has been a topic of interest for many years. Of the many psychiatric disorders, depressive states have been the conditions most strongly associated with this type of mortality (1). Numerous studies have shown a statistical association between depressive disorder or depressive symptoms and subsequent mortality (2-4), but a minority of reports have not confirmed this connection (5, 6).

Proving a causal relation between depression and mortality can be complicated by the confounding effects of factors such as age, sex, social class, and medical (physical) comorbidity (7). Some investigators of depression in the elderly (8, 9) have suggested an interaction between depression and physical illness, so that the effect of depression on mortality may be seen only among patients with physical comorbidity. On the other hand, an association between depression and

mortality has been described even after the data are controlled for the effect of physical comorbidity (2, 7).

A particular concern is whether psychiatric comorbidity alters the survival rate following the onset of physical illness. In a preliminary exploration of this subject (10), we noted that Australian stroke patients with depressive disorder were more than seven times more likely to die over a 15-month follow-up period than nondepressed patients observed for the same amount of time. In addition, we recently completed a 10-year follow-up of a consecutive series of 103 stroke patients examined initially in Baltimore in 1980. Although we previously reported on the results of a 2-year longitudinal study of these patients (11), this report, for the first time, describes the 10-year follow-up data from this group and examines whether depression is associated with mortality.

METHOD

A detailed description of the method of patient selection is available in our previous publication (11). In brief, the study group consisted of a consecutive series of 103 patients admitted to a university hospital stroke unit between 1979 and 1981 with either thromboembolic cerebral infarction or intracerebral hemorrhage. Patients were seen during the acute phase of recovery from stroke (1-3 weeks; mean=11.0 days after the stroke). Informed consent was obtained in all cases.

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Background demographic data were obtained by interview of the patient (plus a relative or friend) and review of the medical chart. Social class was determined using the method of Hollingshead and Redlich (12). Patients were categorized as users or nonusers of alcohol before the stroke (information about the intensity of alcohol use was not available). Neurological examination and diagnosis were done by the attending neurologist using a standardized format and criteria devised for the national pilot Stroke Data Bank (13).

A detailed lesion analysis was performed for a subgroup of 41 patients with no past cerebrovascular disease who had visible single lesions on computed tomography (CT) scans that corresponded to the clinical signs. All CT scans were made with the same slice thickness and angle to the canthomeatal line with the same scanner. Lesion volume (as a percentage of total brain volume) was estimated by measuring the largest cross-sectional area of the lesion and dividing by the area of the brain measured at the slice passing through the body of the lateral ventricles (14).

The Present State Examination (PSE) (15), a semistructured quantitative psychiatric interview, was used to elicit symptoms and signs of mood disorder. Diagnoses of major and minor (dysthymic) depression were made according to DSM-III criteria (excluding the duration criteria). Severity of depressive symptoms was quantified with the Hamilton Rating Scale for Depression (16).

Cognitive impairment was measured with the Mini-Mental State examination (17), and activities of daily living were assessed with the Johns Hopkins Functioning Inventory (18). Prestroke social connectedness was measured with the 10-item Social Ties Checklist (19); scores can range from 0 to 10, with higher scores indicating fewer social ties. The patient's general level of social functioning prior to the stroke was determined by use of the Social Functioning Examination (19), a semistructured interview comprising 28 items. The validity and reliability of the Social Functioning Examination have been described previously (19); scores can range from 0.0 to 1.0, and higher scores indicate poorer prestroke social functioning. Medical comorbidity (serious medical conditions other than the index stroke) was assessed for each patient from interview and from the medical chart. The type of physical illness and the number of illnesses per patient were recorded. The most prevalent conditions were hypertension, coronary artery disease, prior stroke, diabetes, and pulmonary disease.

Follow-up of the patients was undertaken in early 1990 by searching the records of the hospital and the Stroke Data Bank as well as contacting patients or their relatives. In all cases in which death had occurred, an inquiry was made as to whether the death might have been due to unnatural causes (suicide, accidents). Unfortunately, reliable data on cause of death (i.e., autopsy reports) were not available in most cases, although death certificates were obtained for a few patients. The vital status of 12 patients could not be determined.

Data analysis for the 91 patients who were followed up proceeded in two phases. First, patients who had and had not survived were compared on their initial demographic, clinical, social, and psychiatric characteristics. Second, logistic regression was used to examine the relation between depression and mortality, controlling for variables that might potentially confound this relationship. Descriptive statistics (means and standard deviations, medians) were used to summarize data. The chi-square test was used to test for statistically significant associations between nominal variables, and the unpaired *t* test was used to compare groups on continuous measures. The strength of association between predictor variables and mortality was evaluated by using the odds ratio and its 95% confidence interval. The odds ratio (or relative odds) estimates the number of times more likely an outcome (e.g., death) is for a given level of a variable relative to a reference level. Life table analysis (20) was used to calculate probability of survival over time for depressed and nondepressed patients. This analysis takes into account available data from subjects ultimately lost to follow-up. Statistical significance was defined as $p < 0.05$ (two-tailed).

RESULTS

The 12 patients lost to follow-up were compared with the remainder of the subjects on demographic, neurological, social, and psychiatric characteristics. There were no notable differences between the two groups except for age. The patients lost to follow-up were younger (mean age=49.7 years, $SD=12.6$) than the other patients (mean=61.5 years, $SD=12.5$). The frequencies of diagnoses of depression were similar among the two groups: five (42%) of the 12 patients lost to follow-up were depressed, compared with 37 (41%) of the 91 other patients.

The initial demographic, clinical, and psychiatric characteristics of the 91 patients grouped by vital status at follow-up are presented in table 1. At 10-year follow-up, 48 (53%) of the 91 patients had died (none was known to have committed suicide). In a small proportion of cases in which death certificates were available ($N=18$), the ascribed causes of death were as follows: five (28%) from subsequent stroke, eight (44%) from cardiopulmonary arrest, three (16%) from respiratory failure, and two (11%) from other conditions.

Demographic, Clinical, and Social Characteristics and Mortality

The mean age 1–3 weeks after stroke of the patients who were still alive at follow-up was 59.9 years ($SD=13.8$); the mean age of those who had died was 60.9 years ($SD=11.3$). Comparison of the patients who died with those who survived on initial demographic, clinical, and social factors revealed a number of differences. The patients who died had significantly fewer social ties

TABLE 1. Demographic and Clinical Characteristics 1–3 Weeks After Stroke of 91 Patients Grouped According to Their Vital Status at 10-Year Follow-Up

Item	N	Patients Still Alive (N=43)		Patients Who Had Died (N=48)	
		N	%	N	%
Sex					
Female	37	15	41	22	59
Male	54	28	52	26	48
Race					
Black	57	25	44	32	56
White	34	18	53	16	47
Marital status					
Never married	12	3	25	9	75
Married	41	19	46	22	54
Divorced	16	10	63	6	37
Widowed	22	11	50	11	50
Social class ^a					
I–III	22	13	59	9	41
IV	27	14	52	13	48
V	38	15	39	23	61
Handedness					
Right	81	38	47	43	53
Left	10	5	50	5	50
Alcohol use					
Nonuser ^b	46	17	37	29	63
User	45	26	58	19	42
Comorbidity					
None	18	8	44	10	56
Hypertension	16	11	69	5	31
Coronary artery disease	22	7	32	15	68
Prior stroke	16	9	56	7	44
Diabetes	11	4	36	7	64
Pulmonary disease	8	4	50	4	50
Stroke					
Type					
Infarct	80	37	46	43	54
Hemorrhage	11	6	55	5	45
Lesion location					
Right hemisphere	29	16	55	13	45
Left hemisphere	38	15	39	23	61
Brainstem	24	12	50	12	50

^aBecause of missing data, total N=42 for patients still alive and total N=45 for patients who had died.

^bSignificant difference between groups ($\chi^2=3.95$, $df=1$, $p=0.04$).

(mean Social Ties Checklist score=5.2, $SD=1.7$) than the patients who were still alive (mean=4.3, $SD=1.9$) ($t=-2.14$, $df=89$, $p=0.03$). They tended to be more cognitively impaired (mean Mini-Mental State score=20.6, $SD=6.1$) than the patients still living (mean=22.7, $SD=5.8$) ($t=1.72$, $df=89$, $p=0.08$) and were significantly more likely to be nonusers of alcohol before the stroke (table 1). There was a trend for nonsurvivors to have a greater number of other physical conditions at initial assessment (for survivors, mean=1.2, $SD=0.7$; for nonsurvivors, mean=1.5, $SD=1.2$; $t=-1.51$, $df=89$, $p=0.13$), although the frequencies of the different types of conditions were not significantly different for survivors and nonsurvivors ($\chi^2=6.19$, $df=5$, $p=0.28$) (table 1). While there was no difference overall in marital status between the two groups ($\chi^2=3.95$, $df=3$, $p=0.26$), there was a trend for never-married patients to have a higher

TABLE 2. Depression Status 1–3 Weeks After Stroke and Mortality at 10-Year Follow-Up of 91 Patients

Patient Group	N	Patients Still Alive (N=43)		Patients Who Had Died (N=48)	
		N	%	N	%
Nondepressed	54	32	59	22	41
With any depression	37	11	30	26	70
Minor depression	17	5	29	12	71
Major depression	20	6	30	14	70

mortality rate than ever-married patients ($\chi^2=2.74$, $df=1$, $p=0.09$) (table 1). Otherwise, no other demographic or clinical variable was associated with mortality. There were no significant differences between the two groups in scores on the Johns Hopkins Functioning Inventory (mean=6.1, $SD=6.4$, for the patients still living, and mean=7.8, $SD=6.2$, for those who had died) and the Social Functioning Examination (mean=0.19, $SD=0.11$, and mean=0.22, $SD=0.15$, respectively).

Depression and Mortality

The mean PSE score was 5.4 ($SD=4.0$) for the 54 nondepressed patients, 12.2 ($SD=6.1$) for the 17 patients with minor depression, and 21.3 ($SD=7.4$) for the 20 with major depression. The mean Hamilton depression scale scores for these three patient groups were 4.2 ($SD=2.6$), 7.9 ($SD=4.9$), and 14.0 ($SD=6.2$), respectively.

Depressive status was significantly associated with mortality (table 2). Patients with major or minor depression were more likely to have died over the follow-up period. Since mortality was similar in both depressed subgroups, we combined them to form one group ("depressed") in subsequent analyses. Depressed patients were more than three times as likely to die as nondepressed patients (odds ratio=3.4, 95% confidence interval=1.4–8.4, $p=0.007$). Figure 1 illustrates the different survival curves for the depressed and nondepressed patients. The graph shows a divergence in the probability of survival that began as early as the first or second year after stroke and continued to the fifth year after stroke. Subsequently, the survival curves of the depressed and nondepressed patients were parallel. Severity of psychiatric or depressive symptoms was not associated with mortality. The patients who died did not have significantly higher PSE or Hamilton depression scale scores than the survivors (mean PSE scores=10.0, $SD=7.8$, and 9.4, $SD=8.2$, respectively, $t=-0.37$, $df=89$, $p=0.70$; mean Hamilton depression scores=7.2, $SD=5.4$, and 6.6, $SD=5.6$, respectively, $t=-0.46$, $df=89$, $p=0.64$).

We next examined whether a diagnosis of depression (major and minor combined) was associated with mortality after controlling for other variables in a multiple logistic regression model. Factors that tended to be associated with mortality (Social Ties Checklist and Mini-Mental State scores, alcohol use, medical comorbidity,

and marital status) and those that theoretically might confound the relation between depression and mortality (age, sex, social class, type of stroke, hemispheric lesion location, and Johns Hopkins Functioning Inventory and Social Functioning Examination scores) were included in the model. After all of these variables were controlled for, depression remained associated with mortality (adjusted odds ratio=3.7, 95% confidence interval=1.1–12.2, $p=0.03$).

Depression and fewer social ties had an additive effect on mortality. Patients who were depressed and also had few social ties (score above the median on the Social Ties Checklist) had the highest mortality rate (92%, 12 of 13), those who either were depressed or had few social ties had intermediate mortality rates (58%, 14 of 24, and 47%, seven of 15, respectively), and the patients who were not depressed and had many social ties (score at or below the median on the Social Ties Checklist) had the lowest mortality rate (38%, 15 of 39).

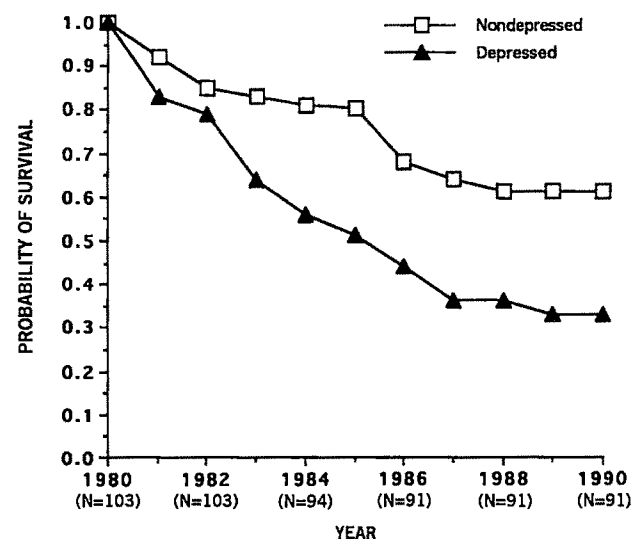
Interactions between depression status and other independent variables were then explored (an interaction was defined as when depression had a strong association with mortality within one level but not within another level of another variable). The association between depression and mortality was significant among patients with good prestroke social functioning (at or below the median score on the Social Functioning Examination) (odds ratio=9.5, 95% confidence interval=2.2–41.1, $p=0.003$) but not significant among patients with poor prestroke social functioning (odds ratio=1.4, 95% confidence interval=0.4–4.6, $p=0.63$). This association was significant among patients who used alcohol before stroke (odds ratio=9.1, 95% confidence interval=2.3–35.9, $p=0.002$) but not significant among nonusers of alcohol (odds ratio=1.49, 95% confidence interval=0.4–5.1, $p=0.53$). Finally, the association between depression and mortality was significant among patients with major medical comorbidity (those with coronary artery disease, prior stroke, diabetes, and pulmonary disorders) (odds ratio=6.8, 95% confidence interval=1.9–24.3, $p=0.003$) but not significant among patients without major medical comorbidity (odds ratio=1.5, 95% confidence interval=0.4–6.0, $p=0.56$).

Lesion Characteristics and Mortality

Of the 41 patients who constituted the subgroup with detailed CT scan data, 28 (68%) were male, 23 (56%) were black, 19 (46%) were married, and most were from the lower socioeconomic classes. Their mean age was 58.6 years ($SD=12.6$). Seventeen patients (41%) had diagnoses of depression (eight major depression, nine minor depression). In this subgroup, depressed patients were three times more likely than nondepressed patients to have died (odds ratio=3.1), but the association did not reach statistical significance (95% confidence interval=0.7–13.8, $p=0.08$).

Among this subgroup of patients, type of stroke (infarct or hemorrhage) and lesion location (right or left

FIGURE 1. Probability of Survival Following Stroke for Depressed and Nondepressed Patients



hemisphere or brainstem) were not associated with mortality ($\chi^2=0.01$, $df=1$, $p=0.94$, and $\chi^2=0.42$, $df=2$, $p=0.81$, respectively). Lesion volume was strongly associated with both mortality and depression status. Patients who died had over twice the lesion volume of patients who survived (mean=10.3%, $SD=9.0\%$, and mean=4.3%, $SD=4.5\%$, respectively; $t=2.70$, $df=39$, $p=0.01$), and depressed patients had three times the lesion volume of nondepressed patients (12.1%, $SD=9.3\%$, and 3.7%, $SD=3.1\%$, respectively; $t=4.10$, $df=39$, $p=0.0002$). Since lesion volume was related to both depression and mortality, we examined the relation between depression status and mortality after matching depressed and nondepressed patients on volume (within $\pm 5.0\%$). Among matched subjects, depressed patients had a higher mortality rate than nondepressed patients (odds ratio=5.4), although this association did not reach statistical significance (95% confidence interval=0.6–65.0, $p=0.09$).

DISCUSSION

The main finding of this study was that a diagnosis of depression in the acute phase of recovery from stroke was associated with more than threefold greater mortality over a 10-year period of follow-up. The effect of depression remained strong even after we controlled for other variables that might be associated with mortality.

Several methodological issues should be considered. First, although the study group consisted of a series of consecutively admitted patients, the patients may not have been representative of individuals who are not admitted to a hospital or who are too ill or aphasic to be interviewed. Second, the assessment of medical comorbidity was limited to noting the type and number of physical conditions. A more detailed description of the

nature and severity of coexisting medical conditions might have revealed a stronger association between comorbidity and mortality and might have altered the association between depression and mortality in the logistic regression analysis. Third, the diagnosis of depression (major and minor types) was based on a cross-sectional interview a relatively short time after the stroke. Although it is possible that some depressions may have been transient disturbances, our previous work (21) has demonstrated that most major and minor depressions diagnosed in this way have durations of 1 year or more. Fourth, although we found no suicides, the relation between depression and ultimate cause of death could not be determined in this study because of the lack of reliable data on cause of death. Fifth, vital status could not be ascertained for 12 subjects, and this may have influenced our results. We think this is unlikely, however, because the missing patients were generally similar to the rest of the study group in demographic profile (except for age), clinical characteristics, and frequency of depression. As a result of these limitations, our findings must be considered preliminary and await replication.

Despite these caveats, it seems reasonable to conclude that a diagnosis of depression may be an independent risk factor for mortality following stroke. The impact of depression on mortality was first seen within 1–2 years and operated for up to 5 years after stroke. Severity of depressive symptoms was not associated with mortality. This finding may seem surprising at first but is probably explained by the fact that the patients with diagnoses of minor and major depression had equivalent high mortality rates despite quite different severities of depressive symptoms.

The depression-mortality association may have been confounded by lesion volume (large lesion volume was associated with both depression and mortality). However, volume is unlikely to account entirely for this association, since after we matched the depressed and nondepressed patients on volume, the mortality rate among the depressed patients remained greater (although the association was not statistically significant). Lesion volume represents a neuropathological predictor of mortality, which probably reflects different etiologies and severities of cerebrovascular disease, factors known to influence stroke outcome (22).

Being depressed and having fewer social ties were associated with a high death rate among our patients. An association between fewer social supports or resources and mortality has also been reported recently in studies of patients with cardiovascular disease (23, 24). The explanation for the high mortality rate among depressed, socially isolated patients remains unclear, but the absence of caring and supportive relationships may leave depressed patients unprotected from the possible adverse consequences of their state of mood, such as poor compliance with treatment or the initiation or worsening of drug and alcohol abuse. Our data also indicate that patients who were users of alcohol, had serious coexisting physical illness, or had good social

functioning before stroke were at a greater risk of death if they suffered from poststroke depression.

Our study adds to the growing evidence that depressed mood is associated with poorer outcome following stroke (10, 25, 26). A prospective study by Frasure-Smith and Prince (27) showed that among patients with myocardial ischemia, mortality was reduced by treating the emotionally distressed patients with supportive psychotherapy. In the light of this provocative finding, it is possible that effective treatment of depression (which would include providing a supportive therapeutic relationship) may also reduce poststroke mortality. This intriguing possibility awaits further investigation.

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Intelligence and Brain Structure in Normal Individuals

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***Objective:** This study was designed to evaluate the relation between intelligence and a variety of measures of brain structure. **Method:** Magnetic resonance imaging scans were used to measure the volume of the intracranial cavity, cerebral hemispheres, lateral ventricles, temporal lobes, hippocampus, caudate, and cerebellum, as well as the overall volume of gray matter, white matter, and CSF, in 67 healthy, normal volunteers. Intelligence was measured with the Wechsler Adult Intelligence Scale—Revised. **Results:** Full-scale IQ was found to be significantly correlated with intracranial, cerebral, temporal lobe, hippocampal, and cerebellar volume but not with caudate and lateral ventricle volume. There were also significant correlations of full-scale, verbal, and performance IQ with overall gray matter volume but not with white matter or CSF volume. Gender differences were noted in the pattern and number of correlations between the volume of the brain and its subregions and full-scale, verbal, and performance IQ. **Conclusions:** The results suggest that the size of some cerebral structures may account for a significant, but modest, proportion of the variance in human intelligence.*

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Modern neuroimaging techniques offer an unparalleled opportunity to examine the relation between brain structure and brain function in normal individuals. Because it is noninvasive and without significant risk, magnetic resonance imaging (MRI) permits in vivo quantitative measurements of brain size and the size of subregions (e.g., the hippocampus) that are thought to subserve known cognitive functions (e.g., memory).

Controversy has persisted for many years about whether there are significant relationships between size and function in the human brain. Geschwind's seminal work, which indicates that the planum temporale is larger on the left side of the brain, reflecting left hemispheric specialization for language, suggested that valid structure-function relationships might exist (1). Subsequent reports have suggested callosal enlargement in left-handed persons, reflecting a higher degree of interhemispheric transfer as well as possible gender differences in brain structure (2–4). While many studies have been done with post-mortem tissue, more recently investigators have also used MRI to conduct "in vivo autopsies" to study these relationships in healthy, normal individuals, permitting the examination of structure-function relationships independent of the

possible epiphenomena produced by aging, illness, death, or the fixation process (5–8).

The relation between intelligence and brain size has been a particularly intriguing question. Several early reports suggested possible relationships between cranial size and intelligence or educational achievement, but these reports have been questioned for a variety of reasons (9–13). MRI has also been used to examine relationships between cerebral size and educational achievement, with the suggestion that a relationship may occur (14, 15). A recent report indicated a high correlation ($r=0.51$) between IQ and cerebral size (16). This particular report focused on college students, who were designated as having a low IQ (103 or less, Wechsler full-scale score) or a high IQ (130 or more, Wechsler full-scale score), thereby enhancing the probability of finding positive correlations between brain size and IQ.

In this article we report the finding of a similar relationship, also based on MRI, in a larger and more generalizable study group. In addition, we measured the size of brain subregions such as the hippocampus and assessed the total volume of gray matter in the brain, which provides an indirect indicator of neuronal density and dendritic expansion.

METHOD

The study group consisted of 67 healthy, normal volunteers recruited through newspaper advertising. They were screened with a structured interview to assess their

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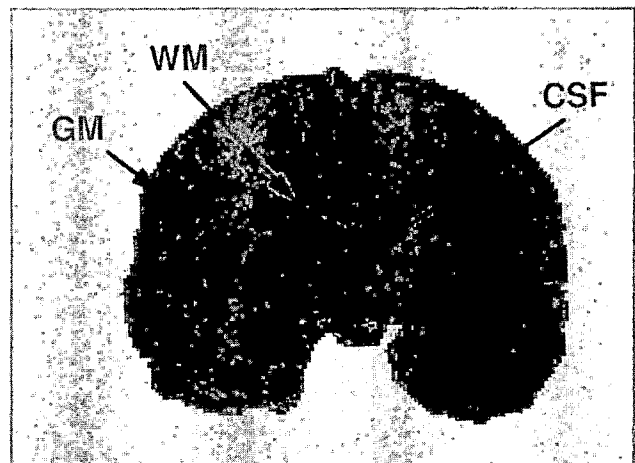
medical and psychiatric histories. Individuals were excluded if they had histories of significant medical, neurological, or psychiatric illness, including alcohol and substance abuse. Thirty-seven subjects were male and 30 were female. Their mean age was 38 years (SD=16). The mean height was 181 cm (SD=7) for the men and 168 cm (SD=6) for the women. Intelligence was assessed with the Wechsler Adult Intelligence Scale—Revised (WAIS-R) (17). The mean full-scale IQ of the group was 116 (SD=14), the mean verbal IQ was 114 (SD=15), and the mean performance IQ was 114 (SD=13). There were no significance differences in IQ between the male and female subjects. The mean educational level of the subjects was 14.5 years (SD=2.5), with no significant difference between the women and the men.

After informed consent was obtained, MRI scans of the subjects were done with a 1.5-tesla General Electric Signa scanner. Slices were acquired in the coronal plane with two separate multiecho sequences. One sequence covered the entire brain, using a 5-mm slice thickness with 2.5-mm gaps (echo time [TE]=30 msec, 90 msec; repetition time [TR]=2,700 msec). A second sequence focused on central brain regions, using 3-mm slices with 1.5-mm gaps (TE=30 msec, 90 msec; TR=3,000 msec), in order to obtain a more precise measurement of the volume of subcortical structures. Data were stored for archival purposes on magnetic tape, transferred to an optical disk, and stored for rapid access on a Qstar optical disk "juke box." Data were analyzed on a Silicon Graphics work station, with locally developed software (18), by a technician blind to the gender and IQ of the subjects.

Two types of MRI measurements were obtained. The first group of measurements assessed the volume of the brain and relevant subregions and was done with a combination of edge detection and manual tracing. Measurements of intracranial, cerebral, cerebellar, and lateral ventricle volume were made from the 5-mm slices, and measurements of the temporal lobes, hippocampus, caudate, and putamen were done from the 3-mm slices. The interrater and test-retest (intrarater) reliabilities of the measurements were assessed and found to be within the acceptable range (e.g., test-retest reliability ranged from a low of 0.60 for the caudate to a high of 0.99 for the cerebrum, and interrater reliability ranged from a low of 0.53 for the hippocampus to a high of 0.99 for the cerebrum).

A second method of measurement involved the use of statistical techniques to classify (segment) brain tissue into one of three categories: gray matter, white matter, and CSF. Methods for performing tissue classification were developed locally and have been described elsewhere (19). Briefly, the method involved identification of "training classes" of the three tissue types, using both the TE=30-msec (proton density) and the TE=90-msec (T_2) images in order to obtain maximally different signal intensities in the various tissue types. Training classes were collected on six samples of each of the three tissue types; these data were then entered into a discriminant function analysis, which was used to classify all pixels in the image into one of the

FIGURE 1. Segmented Image of a Midcoronal Brain Slice^a



^aEach pixel from the MRI scan has been classified as gray matter (GM), white matter (WM), and CSF by a discriminant function analysis; it can be represented visually by a trichotomous (rather than continuous) gray scale. This method permits a quantitative estimate of the total volume of these three tissue types.

three tissue types. A sample segmented image appears in figure 1. The reliability of the method has been assessed and found to be in the acceptable range. Tissue classification has also been validated using phantom studies. Segmentation data were available on three additional subjects, yielding a total study group of 70 for this particular measure.

RESULTS

We found significant correlations between IQ and many of the structures that were measured. In all analyses, height was covaried in order to correct for individual differences in body size. A significant positive correlation was observed between intracranial volume and verbal IQ ($r=0.37$, $N=67$, $p<0.01$), performance IQ ($r=0.27$, $N=67$, $p<0.03$), and full-scale IQ ($r=0.38$, $N=67$, $p<0.01$). As shown in table 1, the three IQ measures were significantly correlated with left and right cerebral volume. Full-scale IQ and verbal IQ were also significantly correlated with left and right temporal and hippocampal volume as well as cerebellar volume. Correlations between performance IQ and these structures were more modest; there were significant correlations with right temporal, left hippocampal, and cerebellar volume. No significant correlations with lateral ventricle or caudate volume were noted.

Table 2 shows the relation between the volume of the three tissue types and intelligence. It suggests that the relationships between IQ and brain size reflect greater gray matter volume, as compared to the volume of white matter or CSF. Significant positive correlations were found between gray matter volume and verbal, performance, and full-scale IQ.

We also examined the relation between gender, size of

TABLE 1. Correlations Between IQ and Volumes of Specific Brain Structures in 67 Normal Subjects in a Magnetic Resonance Imaging Study

Structure	Correlation (r) ^a		
	Verbal IQ	Performance IQ	Full-Scale IQ
Cerebrum			
Left	0.35 ^b	0.26 ^c	0.36 ^b
Right	0.37 ^b	0.28 ^c	0.38 ^b
Temporal lobes			
Left	0.34 ^b	0.20	0.33 ^b
Right	0.50 ^b	0.30 ^c	0.46 ^b
Cerebellum	0.38 ^b	0.41 ^b	0.44 ^b
Hippocampus			
Left	0.41 ^b	0.33 ^b	0.42 ^b
Right	0.32 ^b	0.23	0.32 ^b
Caudate			
Left	0.03	-0.01	0.02
Right	0.13	0.06	0.11
Lateral ventricles			
Left	-0.01	0.07	0.02
Right	0.06	0.09	0.08

^aPearson partial correlation coefficient with height partialled.^bp<0.01.^cp<0.05.**TABLE 2. Correlations Between IQ and Volumes of Gray Matter, White Matter, and CSF in 70 Normal Subjects in a Magnetic Resonance Imaging Study^a**

Measure	Correlation (r) ^b		
	Gray Matter	White Matter	CSF
Verbal IQ	0.31 ^c	0.12	0.03
Performance IQ	0.32 ^c	0.14	-0.09
Full-scale IQ	0.35 ^c	0.14	-0.02

^aData on three additional subjects were used for these measures.^bPearson partial correlation coefficient with height partialled.^cp<0.01.

brain structures, and verbal, performance, and full-scale IQ. These results are shown in table 3. Both male and female subjects showed statistically significant correlations between full-scale IQ and most measures of brain regions. There were differences between male and female subjects in the pattern of correlations with verbal and performance IQ. The women had significant correlations between verbal IQ and intracranial, cerebellar, left and right cerebral, left and right temporal lobe, and left and right hippocampal volume. On the other hand, correlations with performance IQ were limited to the cerebellum, right temporal lobe, and left hippocampus. The men tended to show stronger correlations between size of brain regions and performance IQ and to have fewer significant correlations overall. Both male and female subjects also had significant correlations of IQ with gray matter volume but not with white matter or CSF volume.

DISCUSSION

These results suggest that there is a modest but statistically significant relation between intelligence, as meas-

TABLE 3. Correlations Between IQ and Volumes of Specific Brain Structures Listed Separately for 37 Normal Male Subjects and 30 Normal Female Subjects in a Magnetic Resonance Imaging Study

Structure	Correlation (r) ^a		
	Verbal IQ	Performance IQ	Full-Scale IQ
Cranium			
Male subjects	0.33	0.43 ^b	0.40 ^c
Female subjects	0.43 ^c	0.30	0.44 ^c
Cerebrum			
Left			
Male subjects	0.31	0.41 ^c	0.38 ^c
Female subjects	0.42 ^c	0.29	0.43 ^c
Right			
Male subjects	0.31	0.41 ^c	0.39 ^c
Female subjects	0.46 ^c	0.33	0.47 ^c
Temporal lobes			
Left			
Male subjects	0.14	0.23	0.20
Female subjects	0.53 ^b	0.26	0.49 ^b
Right			
Male subjects	0.41 ^c	0.30	0.39 ^c
Female subjects	0.56 ^b	0.42 ^c	0.56 ^b
Cerebellum			
Male subjects	0.35 ^c	0.43 ^b	0.40 ^c
Female subjects	0.44 ^c	0.46 ^b	0.51 ^b
Hippocampus			
Left			
Male subjects	0.30	0.34 ^c	0.34 ^c
Female subjects	0.51 ^b	0.40 ^c	0.53 ^b
Right			
Male subjects	0.07	0.17	0.13
Female subjects	0.54 ^b	0.32	0.52 ^b
Caudate			
Left			
Male subjects	0.13	-0.06	0.06
Female subjects	-0.16	0.06	-0.06
Right			
Male subjects	0.23	0.05	0.17
Female subjects	-0.07	0.06	-0.01
Lateral ventricles			
Left			
Male subjects	-0.04	0.14	0.03
Female subjects	0.10	0.14	0.13
Right			
Male subjects	0.02	0.14	0.07
Female subjects	0.21	0.23	0.25

^aPearson partial correlation coefficient with height partialled.^bp<0.01.^cp<0.05.

ured by a standard IQ test, and the volume of brain structures, regions, and tissue that may mediate the efficiency of intellectual function. The larger the brain, the higher the IQ. The same relationships were obtained for other measurements, including intracranial, temporal lobe, hippocampal, cerebellar, and gray matter volume. What are the implications of these findings?

First, the modest nature of the relationships must be emphasized. Significant correlations ranged from 0.26 to 0.56, indicating that between 12% and 31% of the variance can be accounted for by the size of the brain or its subregions. This is a relatively small amount of variance, and we must conclude that although size may be among the factors related to human intelligence, many other factors must also be important. What ac-

counts for the remainder of the variance? This particular study cannot address that question. In all likelihood, however, the answer resides in aspects of brain structure that reflect "quality" rather than "quantity" of brain tissue: complexity of circuitry, dendritic expansion, number of synapses, thickness of myelin, metabolic efficiency, or efficiency of neurotransmitter production, release, and reuptake. Factors such as these would facilitate the speed and efficiency of information transfer within the brain as well as expand its capacity, so that multiple tasks of multiple kinds could be performed simultaneously.

Second, the relationships noted suggest that the greater volume of brain tissue associated with higher intelligence may be of a specific type. That is, among the various brain tissue components measured, only gray matter showed a significant positive correlation with intelligence. The greater volume of gray matter can be postulated to reflect a greater number of nerve cell bodies and dendritic expansion; a greater number of neuronal connections presumably enhances the efficiency of computational processing in the brain. Our current methods do not permit us to determine whether gray matter is differentially greater in specific cortical regions or whether the greater volume of gray matter is generalized throughout the cortex. The temporal lobe was the only cortical subregion that was measured; it was selected for measurement because it has clearly identifiable boundaries on coronal MRI scans (unlike frontal, parietal, or occipital regions). Newer methods being developed for reconstructing and visualizing MRI scans, which permit identification of sulcal landmarks on the surface of the brain, will facilitate subsequent studies to determine whether gray matter is selectively greater in particular brain regions (20, 21).

Although both intracranial volume and left and right cerebral size showed significant positive correlations with IQ, subsequent analyses indicated that these greater volumes could be accounted for by selectively greater volumes in brain regions thought to be more closely related to "higher" cognitive functions. Within subcortical brain regions, there was a differentiation between subcortical structures that subserve functions such as memory or language and those that subserve habit formation, motoric function, or emotional function (22–24). That is, a significant correlation was observed between intelligence and hippocampal volume, but not between IQ and the volume of the caudate (22). An IQ test such as the WAIS-R measures multiple domains, including subtests of general information and vocabulary and a variety of others that involve information storage and retrieval. Greater hippocampal size is consistent with an association between intelligence and a greater capacity for information storage and retrieval. In addition, strong positive correlations were noted between cerebellar volume and all three IQ measures. While this association may be seen as counterintuitive, it is in fact consistent with evidence from both neuroanatomical and neuroimaging studies which suggests that the higher phylogenetic level of the

human brain has produced massive expansion in cerebellar size and the development of new and more complex circuitry involved in many "higher" intellectual functions, such as learning, planning, and processing language (25–28).

Can this study tell us anything about how or why high IQ is modestly correlated with greater size of some specific brain structures? At best, it only yields some interesting suggestions but no definitive answers. The correlation between greater intracranial volume and higher IQ suggests, but does not prove, that some factors influencing intelligence and brain size operate relatively early in human development. Head size in human beings is influenced by brain growth; it is largely determined by the second year of life, when the sutures in the skull close, and is nearly complete by the sixth year (29, 30). After the sutures close, skull growth occurs by modeling, which is a relatively slow growth process. Thus, one might infer that the factors that influence head and brain growth and intelligence are in operation quite early. This study does not point to what these factors might be. They include genetic influences, nutrition, environmental stimulation, general health, and a wide range of other factors. This study does not permit any specific inferences concerning the influence of nature versus nurture on either brain growth or intelligence.

What does this study tell us about the relation between gender differences, intelligence, and the size of cerebral structures? The results are consistent with several previous observations, some of which remain controversial: that sex hormones influence brain development (31), that gender differences in brain morphology occur and may be related to cognitive function and behavior (4, 32–34), and that women as a group may have relatively greater skills in tasks that draw on verbal fluency and retrieval, while men as a group have relatively greater skills in the visuospatial domain. Since measures of brain structure were corrected for height, the differential pattern and larger number of the correlations observed in women than in men cannot be explained on the basis of body size. They appear to reflect an interaction between gender differences, brain development and morphology, and cognitive function. This interaction requires further study.

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Trends in Research in Two General Psychiatric Journals in 1969–1990: Research on Research

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Dean Blackwood, B.A., and Thomas Dial, Ph.D.

***Objective:** The authors describe the characteristics of psychiatric research over the past two decades as captured in the articles published by two general psychiatric journals. **Method:** A total of 1,236 articles were drawn from The American Journal of Psychiatry and Archives of General Psychiatry for October through September 1969–1970, 1979–1980, and 1989–1990. Articles were assigned to one of five categories. Research articles were then further categorized as to methodological approach and field of research as well as specific topic areas. Funding sources listed for each research article were also indexed. **Results:** Over time and in both journals, the percentage and number of research articles have risen, with a concomitant reduction in case reports, opinion papers, and “other” articles. Categories of research design were fairly consistent across time and in both journals. Percentages of articles on specific fields and topics indicated an increasing emphasis on biological studies, especially those in clinical psychobiology, as well as a sharp move away from general categories to a more disorder-specific orientation. Reporting of funding sources has substantially increased. **Conclusions:** The large proportion of research articles published in these two important general psychiatric journals reflects editorial policies, changing audience expectations, and the availability of new research tools. Systematic analysis of trends in psychiatric research and other forms of research on research can be useful approaches to assessing the growth and utilization of knowledge in the field, to planning how to most effectively use limited research resources, and to increasing public support for research.*

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Over the past several decades there have been dramatic changes in the practice of psychiatry that have been paralleled by equally dramatic shifts in research activities undertaken by the profession. There has not, however, been substantial effort to systematically portray the nature of those changes in the psychiatric research enterprise (1). The purpose of this paper is to describe the characteristics of psychiatric research as captured in the articles published by two important general psychiatric journals.

There are multiple approaches to characterizing psychiatric research (2). One approach is to investigate the sources of funding for research on mental and addictive disorders (3). Although this approach provides useful information about the types of institutions that provide funding, their fields of interest, and potential opportu-

nities for support and collaboration, it does not capture information about research that is not funded, nor does it provide much understanding of what is being funded. Furthermore, variations in terminology (e.g., “psychiatric,” “mental illness”) make it difficult to assess trends in research funding over time.

Another approach is to document the research activities in academic departments (4). This approach is helpful in assessing the extent of research activity in academic departments and factors associated with higher or lower levels of research activity, but it does not provide information on the products of that activity. There is also difficulty in assessing trends because of the lack of past similar analyses and the problems of retrospective assessment.

Yet another approach is to quantify information about publications in the scientific literature. The published research literature provides an open and concrete composite picture of research in psychiatry. It exists as a historical record that can be evaluated over time to assess trends. Surprisingly, since the major overview study conducted by Brodie and Sabshin in 1973 (1), there has not been a systematic assessment of the re-

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search literature in psychiatry. As part of an effort to acquaint administrators with issues in mental health research and classified abstracts included in a year-book/annual review, Keill (5) provided an examination of difficulties in attempts to classify research. There have been general review articles, literature reviews of specific fields or topics within psychiatry, and annual review volumes published. With rare exceptions (6), these did not typically use a standardized, systematic method for characterizing the range of publications in a given field. Meta-analytic studies are typically limited to summarizing studies by aggregating data on a highly specific phenomenon that can be reduced to an "effect size." In other disciplines and specialties, notably internal medicine, there have been several attempts to evaluate the literature in a systematic and quantitative way (7–11). Most of these studies have involved an assessment of the quality or methods of research reports or statistical issues rather than a description of trends in topical areas or fields.

The present study is an attempt to assess how the published literature in psychiatry has changed by examining articles published during one year in each decade from 1969 to 1990 in two major psychiatric journals. We examined the type of articles published in these journals and the extent to which they might be classified as research, the fields and topics of the research articles, the design of the research studies, and the sources of funding for the research described.

METHOD

We limited our review to articles published in two journals: *The American Journal of Psychiatry* and *Archives of General Psychiatry*. These selections reflect our desire to report on general psychiatric research. For each of the journals, assuming that a year would be representative of the entire decade, we covered a 1-year period in each of the last three decades. The articles were drawn from the months of October through September 1969–1970, 1979–1980, and 1989–1990.

For the purpose of our study, we defined research articles as those which presented original, firsthand data collected in a systematic fashion and which included some clearly delineated methodology. Articles that did not fall under this classification were sorted into one of four other categories: 1) review—an examination of previously published literature, including a synthesis of existing ideas, of which the authors were not the primary source, 2) case report—a presentation of 10 or fewer case reports that contained no summary statistics, 3) opinion—a clearly marked or readily identifiable editorial or commentary, and 4) other—a review other than of research (e.g., an opinion-like essay, not clearly labeled as an editorial, that might include some partial review of previously published literature or descriptions of innovative hospital programs).

Articles that were categorized as research were then filtered through a series of additional classification

schemes, each with specific rules and instructions. The first classification was based on type of methodological design. Feinstein (7) reviewed and described the "research architecture" found in general medical journals. Noting the lack of uniformity in research techniques, Feinstein established no fewer than 17 discrete types of research to account for the wide diversity in the literature. Drawing from Feinstein's work, Fletcher and Fletcher (8) investigated the frequency with which particular research methodologies were used in general medical journal articles. These authors collapsed those categories into more general classes. Following suit, we delineated four general categories of methodological approaches to research: 1) cross-sectional—all measurements of a given subject are taken at only one point in time, 2) case-control/retrospective—subjects are pursued backward in time from effects to causes, 3) cohort/prospective—longitudinal studies in which subjects are followed forward in time and the independent variable does not involve systematic intervention on the part of the investigators, and 4) clinical trial—a type of cohort/prospective study in which randomly selected subjects experience interventions imposed by investigators for the purposes of the study.

We then identified the field of the research to which the article related: basic biological sciences, behavioral/cognitive science, social science, clinical psychobiology, diagnosis/nosology, epidemiology, psychopharmacology, psychosocial treatments, and health/mental health services.

The articles were classified into 13 specific topic categories: schizophrenia/psychotic disorders, affective disorders, anxiety/stress-related disorders, personality disorders, childhood/adolescent mental disorders, eating disorders, alcoholism/substance abuse, basic brain/behavioral science, consultation-liaison psychiatry/behavioral medicine, general/multiple disorders, general policy issues, personality disorders, and geriatric/organic disorders. Also indexed for each article were the source or sources of funding for the study as identified by the authors.

After an intensive training period during which three of us (H.A.P., B.H., and D.B.) rated several dozen articles and then discussed our ratings and refined the categories, one of two raters (B.H. or D.B.) rated each article. If the raters identified a problem in assessing any article, it was discussed among the three of us. An evaluation of the reliability of several articles rated by both raters that required a judgment, such as type of article, methodology, field, and topic, showed an overall agreement rate of 85%.

RESULTS

We reviewed 1,236 articles in the 1969–1970, 1979–1980, and 1989–1990 time periods. The overall findings regarding the categorization of these articles are presented in table 1. In 1989–1990, more than 80% of all articles published in the two journals were research

TABLE 1. Types of Articles Published in *Archives of General Psychiatry* and *The American Journal of Psychiatry* in Three 1-Year Periods

Type of Article	1969–1970		1979–1980		1989–1990	
	N	%	N	%	N	%
Research ^a						
<i>Archives</i>	99	65.1	129	89.6	113	92.6
<i>American Journal</i>	96	40.3	172	51.8	192	77.4
Total	195	50.0	301	63.2	305	82.4
Review ^b						
<i>Archives</i>	13	8.6	7	4.9	4	3.3
<i>American Journal</i>	30	12.6	22	6.6	29	11.7
Total	43	11.0	29	6.1	33	8.9
Opinion ^c						
<i>Archives</i>	3	2.0	1	0.7	3	2.5
<i>American Journal</i>	32	13.4	13	3.9	12	4.8
Total	35	9.0	14	2.9	15	4.1
Case report ^d						
<i>Archives</i>	6	3.9	5	3.5	1	0.8
<i>American Journal</i>	15	6.3	78	23.5	8	3.2
Total	21	5.4	83	17.4	9	2.4
Other ^e						
<i>Archives</i>	31	20.4	2	1.4	1	0.8
<i>American Journal</i>	65	27.3	47	14.2	7	2.8
Total	96	24.6	49	10.3	8	2.2
Total						
<i>Archives</i>	152		144		122	
<i>American Journal</i>	238		332		248	
Total	390		476		370	

^aThe difference among years was significant ($\chi^2=88.40$, $df=2$, $p<0.001$).

^bThe difference among years was significant ($\chi^2=6.83$, $df=2$, $p<0.05$).

^cThe difference among years was significant ($\chi^2=17.25$, $df=2$, $p<0.001$).

^dThe difference among years was significant ($\chi^2=66.11$, $df=2$, $p<0.001$).

^eThe difference among years was significant ($\chi^2=91.35$, $df=2$, $p<0.001$).

articles. This represents a substantial rise over 1969–1970, when only half of the articles reviewed were research articles. In addition, the absolute number of research articles has risen dramatically, from 195 to 305. There has been a related reduction in case reports, opinion articles, and “other” articles. Review articles have remained stable. The significant reduction in case reports between 1979–1980 and 1989–1990 represents a change in the editorial policy of *The American Journal of Psychiatry*, which in 1978 announced that it would no longer print single case reports as articles but instead would include them in letters to the editor.

There are also important differences between the two journals that were more pronounced in earlier years. For instance, *Archives* was more likely to publish research articles in the 1970s, and the *American Journal* published substantially more articles overall and proportionally more case reports and opinion articles. In 1989–1990, *Archives* maintained a higher percentage of research articles—92% compared with 77% of the articles in the *American Journal*—and the *American Journal* had a larger proportion of review or opinion articles than did *Archives*.

With respect to design (table 2), cross-sectional studies and clinical trials made up more than 75% of all the articles in both journals in 1989–1990. Over time there has been considerable stability in design categories, with some rise in retrospective studies. There is a remarkable

TABLE 2. Study Designs of Research Articles Published in *Archives of General Psychiatry* and *The American Journal of Psychiatry* in Three 1-Year Periods

Design	1969–1970		1979–1980		1989–1990	
	N	%	N	%	N	%
Cross-sectional						
<i>Archives</i>	54	54.5	70	54.3	57	50.4
<i>American Journal</i>	54	56.3	95	55.2	91	47.4
Total	108	55.4	165	54.8	148	48.5
Case-control/retrospective ^a						
<i>Archives</i>	5	5.1	5	3.9	4	3.5
<i>American Journal</i>	3	3.1	3	1.7	25	13.0
Total	8	4.1	8	2.7	29	9.5
Cohort/prospective						
<i>Archives</i>	9	9.1	15	11.6	19	16.8
<i>American Journal</i>	9	9.4	24	14.0	30	15.6
Total	18	9.2	39	13.0	49	16.1
Clinical trial						
<i>Archives</i>	31	31.3	39	30.2	33	29.2
<i>American Journal</i>	30	31.3	50	29.1	46	24.0
Total	61	31.3	89	29.6	79	25.9
Total						
<i>Archives</i>	99		129		113	
<i>American Journal</i>	96		172		192	
Total	195		301		305	

^aThe difference among years was significant ($\chi^2=14.52$, $df=2$, $p<0.001$).

similarity in the distribution of designs between the two journals, both over time and in 1989–1990.

Table 3, depicting the fields associated with published articles, makes clear that psychiatric research published in these journals remains primarily clinical and that only a small number of basic science articles are published. In 1989–1990, one-quarter of the articles were in areas of clinical psychobiology and another quarter dealt with treatment research topics (psychopharmacology, psychosocial treatments, and health/mental health services). Approximately one-fifth were in diagnosis/nosology. There was significant growth in research on clinical psychobiology during the 1970s and a more modest increase in psychopharmacology-related articles. The 1980s saw an increase in articles on epidemiology and diagnosis/nosology as well as a continuing rise of articles related to clinical psychobiology. This was accompanied by drops in research articles on behavioral/cognitive science and health/mental health services.

There are also apparent differences between the two journals. The *American Journal* showed a somewhat greater representation of social science, diagnosis/nosology, and health/mental health services research articles, and *Archives* had a greater concentration in clinical psychobiology, with more than double the proportional representation in that field.

The primary topics of the articles, as represented in table 4, were schizophrenia/psychotic disorders, affective disorders, and anxiety/stress-related disorders. Almost three-fifths of the articles were in these categories; the remainder were spread out across a wide array of topics. Regarding changes in topics over time, perhaps what is most striking is the move away from general

TABLE 3. Scientific Fields of Research Articles Published in *Archives of General Psychiatry* and *The American Journal of Psychiatry* in Three 1-Year Periods

Field	1969–1970		1979–1980		1989–1990	
	N	%	N	%	N	%
Basic biological sciences						
<i>Archives</i>	2	2.0	3	2.3	1	0.9
<i>American Journal</i>	3	3.1	2	1.2	2	1.0
Total	5	2.6	5	1.7	3	1.0
Behavioral/cognitive science ^a						
<i>Archives</i>	20	20.2	5	3.9	4	3.6
<i>American Journal</i>	6	6.3	12	7.0	4	2.1
Total	26	13.3	17	5.6	8	2.6
Social science						
<i>Archives</i>	11	11.1	14	10.9	7	6.3
<i>American Journal</i>	14	14.6	21	12.2	24	12.5
Total	25	12.8	35	11.6	31	10.2
Clinical psychobiology ^b						
<i>Archives</i>	21	21.2	32	24.8	43	38.4
<i>American Journal</i>	7	7.3	29	16.9	35	18.2
Total	28	14.4	61	20.3	78	25.7
Diagnosis/nosology						
<i>Archives</i>	18	18.2	21	16.3	16	14.3
<i>American Journal</i>	13	13.5	25	14.5	50	26.0
Total	31	15.9	46	15.3	66	21.7
Epidemiology ^c						
<i>Archives</i>	4	4.0	9	7.0	15	13.4
<i>American Journal</i>	8	8.3	11	6.4	21	10.9
Total	12	6.2	20	6.6	36	11.8
Psychopharmacology						
<i>Archives</i>	10	10.1	24	18.6	19	17.0
<i>American Journal</i>	19	19.8	32	18.6	33	17.2
Total	29	14.9	56	18.6	52	17.1
Psychosocial treatments						
<i>Archives</i>	1	1.0	7	5.4	4	3.6
<i>American Journal</i>	5	5.2	2	1.2	7	3.6
Total	6	3.1	9	3.0	11	3.6
Health/mental health services ^d						
<i>Archives</i>	12	12.1	14	10.9	3	2.7
<i>American Journal</i>	21	21.9	38	22.1	16	8.3
Total	33	16.9	52	17.3	19	6.3
Total						
<i>Archives</i>	99		129		112 ^e	
<i>American Journal</i>	96		172		192	
Total	195		301		305	

^aThe difference among years was significant ($\chi^2=23.22$, $df=2$, $p<0.001$).

^bThe difference among years was significant ($\chi^2=9.29$, $df=2$, $p<0.01$).

^cThe difference among years was significant ($\chi^2=7.08$, $df=2$, $p<0.05$).

^dThe difference among years was significant ($\chi^2=19.77$, $df=2$, $p<0.001$).

^eOne *Archives* article is not accounted for in this analysis.

categories toward much more disorder-specific classifications. In 1969–1970 there was a predominance of articles exploring general/multiple disorders (16%) and general policy issues (15%), but by 1989–1990 the proportion of articles on these topics had fallen to 6% and 5%, respectively. A more dramatic display of this general-to-specific pattern is shown by examining the particular categories of disorders. Of eight distinct disorder classes, only childhood/adolescent mental disorders did not show an increase from 1969–1970 to 1989–1990. In particular, the 1970s saw a substantial increase in

schizophrenia/psychotic disorders-related articles and the 1980s showed a large increase in the anxiety/stress-related disorders category as well as a smaller increase in personality disorders.

A comparison of the two journals with respect to the primary topics of their research articles indicates that *Archives* had a greater proportion of articles in the three topic areas of affective disorders, schizophrenia/psychotic disorders, and, particularly, anxiety/stress-related disorders. The *American Journal* had a larger proportion of articles related to personality disorders, general policy issues, and geriatric/organic disorders.

Table 5 summarizes the results of the analysis of reported funding of the research articles we examined. Overall, in 1989–1990, one-third of the articles reported no funding source. More than 40% received funding from the National Institute of Mental Health (NIMH) or one of the other two institutes of the Alcohol, Drug Abuse, and Mental Health Administration, and 13% received funding from private foundations. Funding from the Veterans Administration, the National Institutes of Health, and internal institutional sources all hovered around approximately 7%. Surprisingly, only 6% of the articles reported funding from industry sources, although this represents some growth from earlier decades. There has been a significant reduction in the number of articles not reporting a source of funding. Whether this represents a change in reporting, journal policies, or the nature of funding for research activity in psychiatry is unclear. A lack of significant change in the proportion of NIMH or other Alcohol, Drug Abuse, and Mental Health Administration support over the past two decades is particularly notable. Moreover, there has been a substantial increase in foreign sources of funding, which may represent an increase in articles from foreign institutions and a somewhat declining American presence (12).

DISCUSSION

The goal of this study is to provide a portrait of the types of scientific information to which psychiatrists have been exposed in two important general psychiatric journals over the past two decades. It does not provide a full picture of all the information that psychiatrists receive from all sources. Psychiatrists are inundated with a plethora of publications, from newsletters and pharmaceutical industry materials to other general and subspecialty psychiatric journals. The study also does not provide a picture of actual research activity in psychiatry. Much research is published in other specialty and subspecialty journals or is not published at all. There are, obviously, other general psychiatric journals, both in the United States and abroad, that publish the results of research conducted in the United States. Evaluating a broader range of journals would provide a more comprehensive picture of scientific information presented to psychiatrists. This would necessitate achieving a consensus on which journals constitute the

TABLE 4. Primary Topics of Research Articles Published in *Archives of General Psychiatry* and *The American Journal of Psychiatry* in Three 1-Year Periods

Topic	1969–1970		1979–1980		1989–1990	
	N	%	N	%	N	%
Basic brain/behavioral science ^a						
<i>Archives</i>	13	13.1	5	3.9	3	2.7
<i>American Journal</i>	4	4.2	7	4.1	3	1.6
Total	17	8.7	12	4.0	6	2.0
Consultation-liaison psychiatry/ behavioral medicine						
<i>Archives</i>	6	6.1	6	4.7	6	5.4
<i>American Journal</i>	2	2.1	8	4.7	9	4.7
Total	8	4.1	14	4.7	15	4.9
Schizophrenia/psychotic disorders ^b						
<i>Archives</i>	12	12.1	32	24.8	26	23.2
<i>American Journal</i>	7	7.3	29	16.9	31	16.1
Total	19	9.7	61	20.3	57	18.8
Affective disorders						
<i>Archives</i>	19	19.2	28	21.7	27	24.1
<i>American Journal</i>	20	20.8	36	20.9	41	21.4
Total	39	20.0	64	21.3	68	22.4
Anxiety/stress-related disorders ^c						
<i>Archives</i>	2	2.0	6	4.7	24	21.4
<i>American Journal</i>	1	1.0	3	1.7	25	13.0
Total	3	1.5	9	3.0	49	16.1
Personality disorders ^d						
<i>Archives</i>	0	0.0	2	1.6	4	3.6
<i>American Journal</i>	3	3.1	2	1.2	18	9.4
Total	3	1.5	4	1.3	22	7.2
Childhood/adolescent mental disorders ^e						
<i>Archives</i>	12	7.9	9	7.0	2	1.8
<i>American Journal</i>	3	3.1	5	2.9	5	2.6
Total	15	7.7	14	4.7	7	2.3
Geriatric/organic disorders						
<i>Archives</i>	3	3.0	2	1.6	2	1.8
<i>American Journal</i>	0	0.0	5	2.9	10	5.2
Total	3	1.5	7	2.3	12	3.9
Alcoholism/substance abuse						
<i>Archives</i>	6	6.1	11	8.5	5	4.5
<i>American Journal</i>	7	7.3	15	8.7	13	6.8
Total	13	6.7	26	8.6	18	5.9
Eating disorders						
<i>Archives</i>	1	1.0	2	1.6	3	2.7
<i>American Journal</i>	1	1.0	4	2.3	7	3.6
Total	2	1.0	6	2.0	10	3.3
General/multiple disorders ^f						
<i>Archives</i>	9	9.1	7	5.4	2	1.8
<i>American Journal</i>	22	22.9	15	8.7	16	8.3
Total	31	15.9	22	7.3	18	5.9
General policy issues ^g						
<i>Archives</i>	13	13.1	15	11.6	4	3.6
<i>American Journal</i>	16	16.7	26	15.1	11	5.7
Total	29	14.9	41	13.6	15	4.9
Other ^h						
<i>Archives</i>	3	3.0	4	3.1	4	3.6
<i>American Journal</i>	10	10.4	17	9.9	3	1.6
Total	13	6.7	21	7.0	7	2.3
Total						
<i>Archives</i>	99		129		112 ⁱ	
<i>American Journal</i>	96		172		192	
Total	195		301		304	

^aThe difference among years was significant ($\chi^2=13.09$, $df=2$, $p<0.01$).^bThe difference among years was significant ($\chi^2=10.14$, $df=2$, $p<0.01$).^cThe difference among years was significant ($\chi^2=50.57$, $df=2$, $p<0.001$).^dThe difference among years was significant ($\chi^2=18.32$, $df=2$, $p<0.001$).^eThe difference among years was significant ($\chi^2=8.06$, $df=2$, $p<0.05$).^fThe difference among years was significant ($\chi^2=16.08$, $df=2$, $p<0.001$).^gThe difference among years was significant ($\chi^2=16.92$, $df=2$, $p<0.001$).^hThe difference among years was significant ($\chi^2=8.06$, $df=2$, $p<0.05$).ⁱOne *Archives* article is not accounted for in this analysis.

TABLE 5. Funding Sources Reported in Research Published in *Archives of General Psychiatry* and *The American Journal of Psychiatry* in Three 1-Year Periods

Source of Funding ^a	1969–1970 (N=195)		1979–1980 (N=301)		1989–1990 (N=305)	
	N	%	N	%	N	%
National Institute of Mental Health	74	37.9	98	32.6	121	39.7
National Institute on Drug Abuse ^b	0	0.0	11	3.7	6	2.0
National Institute on Alcohol and Alcoholism	0	0.0	3	1.0	3	1.0
National Institutes of Health	8	4.1	15	5.0	22	7.2
Veterans Administration ^c	1	0.5	21	7.0	21	6.9
National Science Foundation	1	0.5	0	0.0	1	0.3
Other federal	12	6.2	11	3.7	15	4.9
State	8	4.1	5	1.7	10	3.3
Industry ^d	4	2.1	5	1.7	17	5.6
Internal institutional	8	4.1	23	7.6	23	7.5
Private foundation	16	8.2	35	11.6	40	13.1
Other/foreign ^e	2	1.0	9	3.0	26	8.5
None ^f	94	48.2	137	45.5	100	32.8

^aSome articles listed more than one source of funding.

^bThe difference among years was significant ($\chi^2=8.40$, $df=2$, $p<0.05$).

^cThe difference among years was significant ($\chi^2=11.96$, $df=2$, $p<0.01$).

^dThe difference among years was significant ($\chi^2=8.56$, $df=2$, $p<0.05$).

^eThe difference among years was significant ($\chi^2=18.09$, $df=2$, $p<0.001$).

^fThe difference among years was significant ($\chi^2=13.28$, $df=2$, $p<0.01$).

“psychiatric literature,” which is beyond the scope of this study. By concentrating on the two American general psychiatric journals that have the largest circulations we hoped to achieve an approximation of that more comprehensive view.

One final caveat: although we did assess the designs of the studies published, no formal assessment of the quality of the papers was conducted. All articles published in these journals have undergone rigorous peer review, and far more manuscripts were submitted than were ultimately published. The decision about what is published is subject to editorial selection of papers from a large pool of high-quality manuscripts. Although bibliographic assessment has suggested progressively increasing citation indexes for each of these two journals over time (Evelyn Myers, personal communication), even these quantifiable approaches do not gauge the overall importance of a particular article for the field with regard to ultimate influence on clinical practice and patient care.

Perhaps the most significant finding in this study is the increasing growth—in both proportion and number—of research articles in the two major general psychiatric journals. This represents the continuation (with a somewhat steeper slope) of the trend initially noted by Sabshin and Brodie in 1973 (1) and discussed by Pardes and Pincus in 1980 (13). Although the information communicated to psychiatrists through these journals is now overwhelmingly research-based, the reasons for this dramatic shift are unclear. It is tempting to conclude that there has been a surge in research activity in psychiatry, but that may not portray the whole story. Between 1969 and 1990, the proportion of research articles increased from 50% to 82% and the absolute number increased by more than 50%. During the same period, there was no real growth in NIMH research funding. This lack of growth is reflected in the data indicating that the proportion of re-

search articles acknowledging NIMH funding did not increase significantly.

The findings also suggest other limitations on a potential interpretation of the links between funding and research publication: the extent to which research data were published in these general journals (as contrasted to subspecialty journals) or published at all was not assessed. For example, acknowledgment of pharmaceutical funding increased over the three study periods, but pharmaceutical companies were named as a source of support for only 2% to 6% of the research articles. Other sources estimate much higher rates of pharmaceutical funding overall for biomedical research (14). It is likely that a substantial amount of these funds support preclinical studies unlikely to be published in the clinical literature as well as clinical data that may be published in subspecialty journals. Clinical trials with negative results that were supported by pharmaceutical industry sources may be less likely to be submitted for publication (15). There is also a question as to whether authors have consistently acknowledged all funding sources over time. Nevertheless, a significant drop in the proportion of articles reporting no funding source suggests that it is becoming increasingly difficult to conduct a research study publishable in one of these psychiatric journals without having some specific source of funding.

Another, perhaps more likely, basis for the increasing number and proportion of research studies is the interaction among the editorial decision-making processes, the expectations of journal readers, and the overall context of psychiatry in the latter half of the twentieth century. The role of journal editorial policies is suggested by the differential rate of change between the two journals. *Archives of General Psychiatry* has had the same editor over the three study periods and, starting out

with a relatively high proportion of research studies, increased that proportion more gradually. *The American Journal of Psychiatry*, on the other hand, underwent a change in editor in 1978 and had a much more dramatic shift in the distribution of types of articles between 1980 and 1990. One should not infer that these shifts are simply the result of decisions by any one editor. In fact, changes at all points along the editorial review and decision-making process could have influenced this change, ranging from selection of peer reviewers, instructions given, use of statistical consultants, and selection of members of editorial boards.

Furthermore, there is constant and extensive formal and informal interaction between the editorial processes of a journal and its audience—both those who submit manuscripts to the journal and those who read the published articles. This interaction occurred during the period of time in American psychiatry that has been characterized by a rapid shift from “boundary expansion, the predominance of ideology over science, and . . . demedicalization” (16) to one much more strongly linked to the mainstream of American medicine and the overall biomedical research establishment.

The results with regard to fields and topics also support this increasing sense of boundaries of and within psychiatry. To some extent, this has been fueled by the development of new tools and measures for research. Development of neuroimaging techniques and assays for measurement of neuropeptides, receptor physiology, regional glucose metabolism, and other physiological and structural aspects of the human brain underlie the tremendous growth in the studies of clinical psychobiology. Similarly, the development of DSM-III and its predecessors of research diagnostic criteria, along with other measures of mental states, have enabled the identification and study of more specific mental disorders.

To some extent, the rapid growth and development of the capacity to measure may have kept the distribution of research designs stable over the course of time. All of these new tools can be most immediately applied in cross-sectional studies. The mobility of both investigators and patients, as well as the “publish or perish” mentality in academic settings, may also influence this distribution by reducing the capacity to conduct longitudinal or prospective studies. This was noted by Fletcher and Fletcher (8) in their examination of research design in the general medical literature, which in 1978 showed a reasonably similar distribution to that in the general psychiatry literature. We would hope that these new tools see greater application over the next decade in prospective studies.

In a continuation of the trends noted by Sabshin and Brodie (1) there is a continuing rise in the proportion of biologically oriented articles as well as a drop in the proportion of psychologically and sociologically oriented articles, particularly those on psychological and behavioral precursors and mechanisms associated with particular mental disorders. This may reflect the fact that the surge of new tools and instruments and treat-

ments has largely been in the biological realm and has been applied to patient populations with increasingly discrete diagnoses. It is possible, however, that some of the psychological and sociological articles might have drifted off into other publications and thus are not as accessible to psychiatrists generally.

The drop in the proportion of studies on mental health services may seem at first somewhat surprising, in view of the attention given this field by NIMH programs over the past decade (17). It is likely, however, that what is not captured in the data may be a screening out of very limited studies with a more rudimentary focus on the evaluation of specific programs and a retention of more methodologically sophisticated studies addressing broader issues in health services research and policy.

DSM-III and DSM-III-R have been both a reflection of and a stimulation for the increased focus of research on specific disorders. In the disorder-based groupings the emphasis on affective, schizophrenic, and anxiety disorders has not only been stable over time but is also remarkably consistent with data about the diagnostic distribution of practicing psychiatrists' caseloads (18). With the small reduction in the number of articles on schizophrenia/psychotic disorders between 1980 and 1990, following substantial growth between 1969 and 1980, it would be interesting to see to how the recently introduced NIMH National Plan for Schizophrenia Research (19) will affect the scope and magnitude of research on schizophrenia over the next decade. Research articles on anxiety/stress-related disorders and, to a lesser extent, personality disorders have increased over the past decade. Changes in anxiety/stress-related disorders are most certainly reflective of the elucidation of more specific disorders, as exemplified in DSM-III-R, as well as through the use of techniques of “pharmacological dissection” described by Klein (20). Similarly, the development of new instruments for the assessment of personality disorders and increasing interest in their classification and treatment contribute to resurgence of interest in this area.

Disciplines recently proposed for subspecialty designation in psychiatry (geriatrics, substance abuse, and consultation-liaison psychiatry), as well as the longstanding subspecialty of child psychiatry, have relatively small representation in the general psychiatric research literature. This may be due partially to the existence of subspecialty-specific journals. Childhood/adolescent disorders in particular have received very limited attention in the general psychiatric research literature over the past several decades. This view is reinforced by the Institute of Medicine report on research in child and adolescent mental disorders (21).

CONCLUSIONS

Systematic, quantitative analyses of the literature can provide a useful overview of a field and illuminate changes over time. Augmented with such techniques as citation analysis, these approaches can suggest some

measure of quality assessment. Accordingly, these methods can be "valuable tools in studies of the effectiveness of different mechanisms for the funding of research," as noted by Anderson and Evered (2). The development of standardized classification systems, such as for the categories described here and others, which would be used uniformly by journals and perhaps encoded in a standardized format, would be an enormous step forward in conducting such studies. Furthermore, a better understanding of the larger universe of research activity, both published and unpublished, would be available if journals produced summary data on submissions in a standardized format.

There are, of course, limits to these approaches. The assessment of quality requires more than computerized bibliographic algorithms. Knowledge of clinical applicability and scientific salience—the assessment as to whether "new knowledge" is redundant, trivial, important, or a breakthrough—as well as understanding of its actual effects on clinical practice and patient care—require much more extensive and (to some extent) subjective assessments. Furthermore, simply looking back at the past may not be the ideal means of "appraising the future prospects of emerging areas of research" (2). Given the scarcity of research funds, it is important that there be a more comprehensive and systematic program for evaluation of the process and outcome of scientific activities. It will be necessary to use multiple approaches to scientifically examine research and research training processes, activities and characteristics of scientists, and quantity and quality of research output to assess the most promising and fruitful directions for the field. "Research on research" is needed to make judgments most efficiently and effectively about where to place limited resources. An important side benefit of such an effort would be the demonstration to public policy makers, legislative representatives, and the public that the scientific community takes seriously its responsibilities and accountabilities to society, thus engendering more substantial public support.

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Physician-Assisted Suicide: The Dangers of Legalization

Herbert Hendin, M.D., and Gerald Klerman, M.D.

The authors examine physician-assisted suicide in the light of what is known about suicide and terminal illness, exploring the potential for abuse if legalization occurs. The elderly, those frightened by illness, and the depressed of all ages would be potential victims. The authors discuss the cases that have received public attention as illustrative of these abuses.

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There are situations when helping a terminally ill patient end his or her life seems appropriate. For centuries physicians have helped such patients die. Why should we not protect them and at the same time make it easier for the terminally ill to end their lives by legalizing physician-assisted suicide? The movement to do so represents such a drastic departure from established social policy and medical tradition that it needs to be evaluated in the light of what we now know about suicide and terminal illness.

We know that 95% of those who kill themselves have been shown to have a diagnosable psychiatric illness in the months preceding suicide (1-4). The majority suffer from depression, which can be treated. This is particularly true of the elderly, who are more prone than younger victims to take their lives during the type of acute depressive episode that responds most effectively to modern available treatments (3). Other diagnoses among the suicides include alcoholism, substance abuse, schizophrenia, and panic disorder; treatments are available for all of these illnesses.

Advocates of physician-assisted suicide try to convey the impression that in terminally ill patients the wish to die is totally different from suicidal intent in those without terminal illness. However, like other suicidal individuals, patients who desire an early death during a terminal illness are usually suffering from a treatable

mental illness, most commonly a depressive condition (5). Strikingly, the overwhelming majority of the terminally ill fight for life to the end. Some may voice suicidal thoughts in response to transient depression or severe pain, but these patients usually respond well to treatment for depressive illness and pain medication and are grateful to be alive.

Studies of those who have died by suicide have pointed out the nonrational elements of the wish to die in reaction to serious illness. More individuals, particularly elderly individuals, killed themselves because they feared or *mistakenly* believed they had cancer than killed themselves and actually had cancer (6, 7). In the same vein, preoccupation with suicide is greater in those awaiting the results of tests for HIV antibodies than in those who know that they are HIV positive (8).

Given the advances in our medical knowledge and treatment ability, a thorough psychiatric evaluation for the presence of a treatable disorder may literally make the difference between choosing life or choosing death for patients who express the wish to die or to have assisted suicide. This is not an evaluation that can be made by the average physician unless he or she has had extensive experience with depression and suicide (9).

Even the highly publicized cases that have been put forward by the advocates of legalizing assisted suicide dramatize the dangers and abuses we would face when those who are not qualified to do so evaluate such patients or when we accept at face value a patient's assertion that he or she prefers death. Perhaps the first such case was featured in a front-page story more than a decade ago (10). It concerned a woman who, after being diagnosed as having breast cancer, brought together her friends and her husband (who was a psychologist),

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Dr. Klerman died in April 1992.

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filmed her farewells, and took a lethal overdose. For years the woman had been an advocate of the "right to suicide." Her film became a television documentary, and media stories portrayed her as something of a pioneer. A pioneer for what? Does her story contain a message we wish to send to the thousands of women facing possible breast surgery? The woman was not terminally ill; her cancer was operable. Although her psychologist husband supported her decision and felt it was appropriate, surely he was not the person to evaluate her. Was her choice as rational as everyone claimed?

Suicidal individuals are prone, just as this woman was, to make conditions on life: "I won't live if I lose my breast," "if this person doesn't care for me," "if I don't get this job" or "if I lose my looks, power, prestige, or health." Depression, often precipitated by discovering a cancer, exaggerates the tendency toward rigid thinking, toward seeing problems in black-and-white terms (11).

More recent cases are equally troubling. In the *New England Journal of Medicine*, a physician published the case of a woman whom he helped to commit suicide (12). The woman had a past history of both alcoholism and depression and had recently been diagnosed as having acute leukemia. Her chances of surviving painful chemotherapy and radiation were assessed as one in four. She told her doctor that "she talked to a psychologist she had seen in the past" and implied that the psychologist supported her decision to commit suicide. The physician helped her to implement her decision to end her life. He then published an account of what transpired in an attempt to persuade the medical community of the need for legal sanction for his actions.

The fact that this or any patient may find relief in the prospect of death is not necessarily a sign that the decision is appropriate. Many who are depressed and suicidal appear less depressed after deciding to end their lives. It is coping with the uncertainties of life and death that agitate and depress them. One would need a far more extended examination by someone knowledgeable about suicide to evaluate this woman.

Depression, which is often covert and can coexist with physical illness, is, together with anxiety and the wish to die, often the first reaction to the knowledge of serious illness and possible death. This demoralizing triad can usually be treated by a combination of empathy, psychotherapy, and medication. The decision whether or not to live with illness is likely to be different with such treatment.

The publications of groups like the Hemlock Society, who advocate a more general "right to suicide," make clear that physician-assisted suicide for patients who have less than 6 months to live (as in the recently defeated California and State of Washington proposals) is but a first step in their campaign. Only a small percentage of the people they are trying to reach are terminally ill. The terminally ill, in fact, constitute only a small portion (less than 3%) of the total number of suicides (1, 3, 9). Right-to-suicide groups have been joined in their efforts by well-meaning physicians concerned with the plight of the terminally ill.

Discussions of the right to suicide or the rationality of suicide in particular cases have tended to ignore the potential for abuse were physician-assisted suicide to be legalized. Particularly vulnerable potential victims would be the elderly, those frightened by illness, and the depressed of all ages.

The elderly are often made to feel that their families would prefer that they were gone. Societal sanction for physician-assisted suicide for the terminally ill is likely to encourage family members so inclined to pressure the infirm and the elderly and to collude with uninformed or unscrupulous physicians to provide such deaths. Some advocates of changing social and medical policy toward suicide concede that such abuses are likely to occur but feel that this is a price we should be willing to pay (13).

Those whose terror of illness persuades them that quick death is the best solution may be willing victims of physicians who advocate assisted suicide. A woman in the early stages of Alzheimer's who was fearful of the progress of the disease was seen briefly by Dr. Jack Kevorkian, a retired pathologist in Michigan with a passionate commitment to promoting assisted suicide and the use of his "suicide machine." After a brief contact he decided she was a suitable candidate. He used the machine to help her kill herself. Is he the person who should be making such a determination? No Michigan law prohibits assisted suicide (19 states do not have such laws), but Dr. Kevorkian was admonished by the court not to engage in the practice again. Disregarding the admonition, he subsequently provided machines to two more women who were seriously but not terminally ill (14). They used the machines to kill themselves. Dr. Kevorkian's license to practice medicine has since been "summarily suspended," but a Michigan judge ruled that he could not be prosecuted for murder in the absence of a state law prohibiting assisting a suicide.

Societal sanction for physician-assisted suicide is likely to encourage assisted suicide by nonphysicians, rendering those who are depressed, with or without physical illness, vulnerable to exploitation. Such abuse already exists. For example, a young man gave a depressed young woman he knew a lethal quantity of sleeping pills. He sat with her and fed them to her as she ate ice cream. While she was doing so, he persuaded her that, since she was going to die, she should write out a will leaving him her possessions. He went home and told his roommate what he had done; the roommate called the police and the young woman was saved. The young man went unpunished because he did what he did in a state with no law prohibiting assisted suicide (11).

So-called suicide pacts, often romanticized by the press, provide another example of such abuse. Published case reports confirm our own clinical experience, which indicates that such pacts are usually instances where a man who wishes to end his life coerces a woman into joining him as proof of her love (11, 15, 16). In her book, her taped suicide note, her letters, and conversations with friends, the former wife of Derek Humphry,

the founder of the Hemlock Society, made clear that she was tormented by having actively participated with Humphry in the suicide pact of her parents. Although her 92-year-old father may have been ready to die, she was aware that her 78-year-old mother was not (17-19).

Surely there is a price to be paid for current policy where physicians, patients, and family members must act secretly or may be unwilling to act even in situations where it seems appropriate. The protection of the honorable physician does not now warrant legalizing physician-assisted suicide in a society where the public is relatively uninformed of present abuses involving assisted suicide and the potential for much greater abuses if legalization occurs. It took us several decades to become knowledgeable about when it may be appropriate to withdraw life support systems. We are not close to that point with physician-assisted suicide.

Nor by itself can evaluation of the patient by psychiatrists knowledgeable about suicide, depression, and terminal illness provide us with a simple solution to a complex social problem. Certainly, the individual physician confronted with someone requesting assisted suicide should seek such consultation. There is still too much we do not know about such patients, too much study yet to be done before we could mandate psychiatric evaluation for such patients and define conditions under which assisted suicide would be legal. We are likely to find that those who seek to die in the last days of terminal illness are a quite different population from those whose first response to the knowledge of serious illness is to turn to suicide.

Not all problems are best resolved by a statute. We do not convict or prosecute every case in which someone assists in a suicide, even in states where it is illegal. Given the potential for abuse, however, to give assisted suicide legal sanction is to give a dangerous license.

In some cultures (the Alorese are perhaps the most famous example), when people became seriously ill, they took to their beds, stopped eating, and waited to die. How we deal with illness, age, and decline says a great deal about who and what we are, both as individuals and as a society. The growing number of people living to old age and the increasing incidence of depression in people of all ages present us with a medical challenge. Our efforts should concentrate on providing treatment, relieving pain for the intractably ill, and, in the case of terminal illness, helping the individual come to terms with death.

If those advocating legalization of assisted suicide prevail, it will be a reflection that as a culture we are turning away from efforts to improve our care of the mentally ill, the infirm, and the elderly. Instead, we would be licensing the right to abuse and exploit the fears of the ill and depressed. We would be accepting the view of those who are depressed and suicidal that death is the preferred solution to the problems of illness, age, and depression.

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Magnetic Resonance Imaging of Schizophrenia-Like Psychoses Associated With Cerebral Trauma: Clinicopathological Correlates

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Three patients with schizophrenia-like psychosis and two with schizoaffective-like psychosis who experienced cerebral trauma before the onset of their illness underwent clinical and magnetic resonance imaging evaluation. Each patient with a schizophrenia-like psychosis, but neither of those with a schizoaffective-like psychosis, showed abnormalities confined to or including the left temporal lobe. These observations complement recent findings in schizophrenia.

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The relationship to schizophrenia of the schizophrenia-like organic psychoses has long been recognized as an issue of fundamental importance (1, 2). Recent insights into the pathology of schizophrenia itself have prompted a reevaluation of this relationship and of the dilemma posed by contemporary operational criteria that deem schizophrenia to be present only in the absence of an organic substrate (2). These psychoses are also of relevance to the concept of heterogeneity within schizophrenia, a perspective advocating research into forms of the disorder where particular putative etiological factors appear most relevant. Such research may further our understanding of the basic mechanisms underlying schizophrenia (3).

The schizophrenia-like psychoses associated with cerebral trauma present such an opportunity (1). We have previously used magnetic resonance imaging (MRI) to evaluate a patient with psychosis associated with cerebral trauma (4), and we further explore here

the significance of these psychoses in the light of recent neuroimaging and neuropathological findings in schizophrenia.

METHOD

Records of patients who received care in our hospital, as compiled by their treating psychiatrists, were reviewed to identify those who had an apparent schizophrenia-like illness of putative organic origin. Patients were then selected strictly on the basis that their illness was preceded by clinically significant cerebral trauma (loss of consciousness greater than 4 hours' duration) and that their psychosis occurred in clear consciousness and was otherwise indistinguishable from the corresponding DSM-III-R diagnosis, with the exception of the history of cerebral trauma.

Each patient so identified gave informed consent to participate and was interviewed by a research psychiatrist (P.B.) concerning that episode of head injury and the patient's personal and psychiatric history. The patient's current psychotic symptoms were also evaluated with the Scale for the Assessment of Positive Symptoms and the Scale for the Assessment of Negative Symptoms (5). A relative was also interviewed regarding the patient's cerebral trauma and to ascertain any history of

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TABLE 1. Clinical and MRI Findings in Five Patients With Schizophrenia- or Schizoaffective-Like Psychoses After Cerebral Injury

Patient	Sex	Age (years)			Positive Symptoms ^a	Negative Symptoms ^b	MRI Findings ^c		
		At Interview	At Cerebral Injury	At Onset of Psychosis			Qualitative	Left Ventricle Volume (mL)	Right Ventricle Volume (mL)
Patients with schizophrenia-like psychosis									
1	M	43	25	26	0	4	Left temporal lobe gliosis; left temporal horn dilatation	2.3	2.7
2	F	47	21	40	9	4	Lateral ventricular dilatation (left greater than right); left temporal and parietal lobe atrophy	16.1	7.7
3	M	24	12	19	1	15	Ventricular dilatation, especially left frontal horn; gliosis of frontal lobes (left greater than right) and of left anterior temporal lobe	22.1	15.4
Patients with schizoaffective-like psychosis									
4	M	29	2	18	8	2	No abnormality evident	2.6	2.9
5	M	37	10	21	4	5	No abnormality evident	10.8	9.8

^aScale for the Assessment of Positive Symptoms (5).^bScale for the Assessment of Negative Symptoms (5).^cFor 18 patients with DSM-III-diagnosed schizophrenia with a mean age of 30.2 (SD=5.8) the comparable mean left ventricle volume was 7.3 mL (SD=4.0) and the comparable mean right ventricle volume was 6.1 mL (SD=2.2).

schizophrenia, operationally defined (6), among either first- or second-degree relatives.

Each patient also underwent MRI examination (1.5 T Siemens Magnetom) with sequences described elsewhere (7); the images were evaluated by a MRI neuro-radiologist (J.P.S.) who was blind to the patient's status, and volumetric analysis of the lateral ventricles was performed from contiguous 6-mm sections. Comparisons were made with appropriately age-matched patients with DSM-III-diagnosed schizophrenia from our recent MRI study (7), supplemented by three normal, volunteer comparison subjects (men, aged 26, 27, and 32), all of whom were examined according to identical procedures.

RESULTS

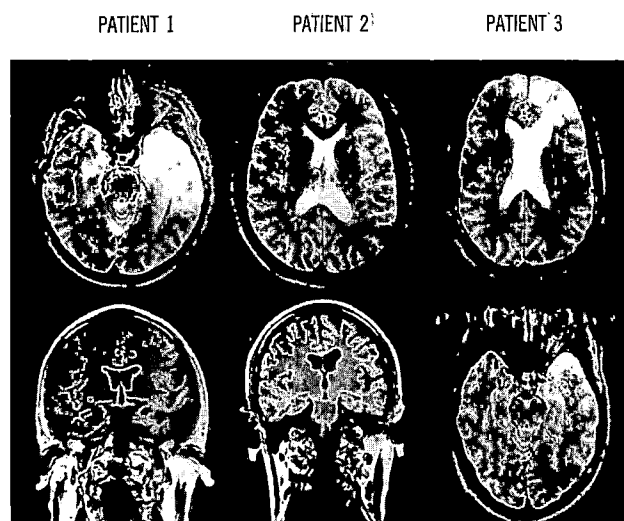
Application of these strict criteria following the extensive record review yielded five patients (three with a schizophrenia-like psychosis and two with a schizoaffective-like psychosis) whose illness was preceded by cerebral trauma (patient 1 from a blow to the head, patients 2, 3, and 5 from road traffic accidents, and patient 4 from a fall from a height). Their clinical and neuroradiological characteristics are given in table 1 and figure 1.

All three of the patients with schizophrenia-like psychosis but neither of the patients with schizoaffective-like psychosis showed abnormalities that included the left temporal lobe and dilatation of the left ventricular system. In patient 1 the only abnormality evident was left temporal lobe gliosis with dilatation of the left temporal horn. No such left temporal lobe abnormalities were evident in any of the comparison subjects.

DISCUSSION

Ventriculomegaly and abnormalities of the temporal lobe region, particularly the left hemisphere, have been described in neuropathological and neuroimaging studies in schizophrenia (7–9). Thus, the pattern and location of MRI findings in our patients 1, 2, and 3, all of whom were right-handed and had a schizophrenia-like psychosis after cerebral trauma, provide further support from an alternative but complementary perspective for left ventricular enlargement and for left temporal lobe dysfunction in schizophrenia. It may also be noteworthy that the pattern of MRI abnormalities for patient 3, whose illness was indistinguishable from the deficit syndrome of schizophrenia, closely resembled a

FIGURE 1. Representative Magnetic Resonance Images of Three Patients With Schizophrenia-Like Psychosis^a



^aFor patient 1, the left upper axial and left lower coronal are shown. For patient 2, the center upper axial and center lower coronal are shown. For patient 3, the right upper axial and right lower axial are shown.

proposed frontal and temporal (amygdalar) basis for the deficit syndrome (10). The extent and consistency of MRI abnormalities in these three patients stand in contrast to the wide variability in the interval between cerebral trauma and psychosis. Although these findings are compelling, their interpretation would be facilitated by further research comparing patients who experienced cerebral trauma and who did or did not develop psychosis subsequently.

Four of our patients had no family history of schizophrenia, but an uncle of patient 1 had himself experienced a head injury before the onset of psychosis. The modest number of patients studied precludes definitive inferences as to genetic risk, but our findings are consistent with the idea that the risk for schizophrenia among relatives of patients with a schizophrenia-like psychosis is considerably lower than the risk in relatives of patients with schizophrenia (1).

The existence of structural and functional brain changes in schizophrenia challenges our concept of this disorder and its current diagnostic boundaries. Such findings blur the previously held sharp distinction between schizophrenia and the apparently more rare schizophrenia-like psychoses. Our patients with schizophrenia-like psychosis after head injury, and the similarity of their MRI findings to neuropathological abnormalities reported in schizophrenia, support the proposition that such psychoses may be but one form of the phenotypical disorder that Bleuler originally called "the group of schizophrenias" (1-3). Furthermore, since these cases may reflect more gross examples of the cerebral dysfunction now believed to be a feature of schizophrenia itself, they question the validity of contemporary operational criteria that preclude this diagnosis in the presence of evident organic disease.

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CSF Homovanillic Acid in Schizotypal Personality Disorder

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CSF concentrations of homovanillic acid (HVA) were measured in 10 patients with schizotypal personality disorder and 14 patients with other personality disorders. The patients with schizotypal personality disorder had higher CSF HVA concentrations than the patients with other personality disorders. Furthermore, the psychotic-like schizotypal symptoms correlated positively with the CSF HVA concentrations. These results suggest a central dopaminergic dysfunction associated with the psychotic-like symptoms of schizotypal personality disorder.
(Am J Psychiatry 1993; 150:149–151)

Schizotypal personality disorder appears to be genetically, biologically, and phenomenologically related to chronic schizophrenia along a continuum of schizophrenia-related disorders (1). Patients with schizotypal personality disorder display psychotic-like symptoms (ideas of reference, cognitive/perceptual distortions, and suspiciousness) analogous to the psychotic symptoms of schizophrenia (1). These psychotic-like symptoms of schizotypal personality disorder are diminished by neuroleptics (2), dopamine receptor antagonists. These considerations raise the possibility that dopamine activity modulates the psychotic-like symptoms of schizotypal personality disorder, perhaps related to an underlying central dopamine dysfunction. Since the CSF concentrations of the dopamine metabolite homovanillic acid (HVA) may reflect in part central dopamine neuronal activity (3, 4), we compared the CSF HVA concentrations in patients with schizotypal personality disorder and in patients with other personality disorders. Preliminary data from this study have been published elsewhere (5).

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METHOD

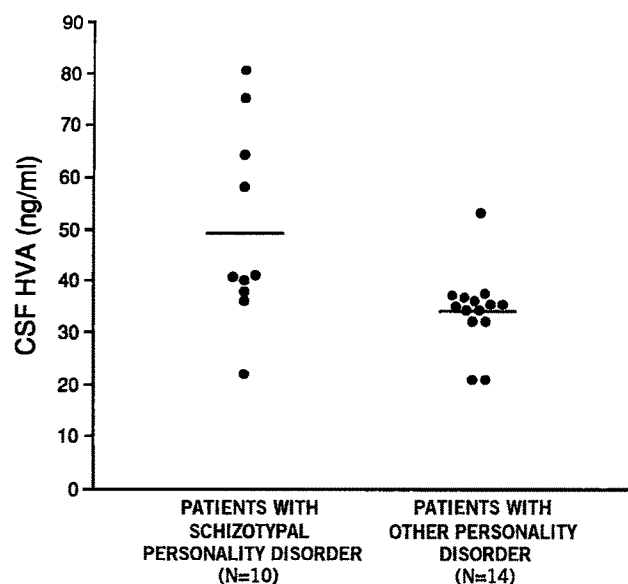
Patients were screened for this protocol as part of an ongoing study of patients with personality disorders from consecutive inpatient and outpatient admissions at two metropolitan medical centers. Patients with any evidence of medical illness according to laboratory or physical examination were excluded from study.

There were 10 patients in the group with schizotypal personality disorder. All were men. Their mean age was 33.6 years (SD=9.2), and their mean height was 171.0 cm (SD=9.9). There were 14 patients in the group with other personality disorders. Twelve were men and two were women. Their mean age was 35.4 years (SD=7.2), and their mean height was 174.8 cm (SD=8.8).

The participating patients granted informed consent for lumbar puncture. Each patient was interviewed by one or two interviewers using the Schedule for Affective Disorders and Schizophrenia (SADS) (in our laboratory, for schizophrenia the interrater reliability kappa is 0.80 and for major affective disorders the interrater reliability kappa is 0.87) and the Structured Interview for the DSM-III Personality Disorders (6), including an interview with an informant close to the patient (in our laboratory, for schizotypal personality disorder the interrater reliability kappa is 0.73). The DSM-III schizotypal personality disorder symptoms of magical thinking, ideas of reference, recurrent illusions, and suspiciousness were considered psychotic-like symptoms. Patients with paranoid and schizoid personality disorders were excluded from the group of patients with other personality disorders because of the possible relationship of these diagnoses to schizophrenia.

Patients meeting current or past DSM-III criteria for any chronic axis I disorder that might confound axis II

FIGURE 1. CSF HVA Concentrations of 10 Patients With Schizotypal Personality Disorder and 14 Patients With Other Personality Disorders^a



^aThe CSF HVA concentrations of the patients with schizotypal personality disorder were higher (mean=49.6 ng/ml, SD=18.9) than those of the patients with other personality disorders (mean=34.4 ng/ml, SD=7.6) ($F=7.51$, $df=1, 22$, $p=0.01$).

assessment were excluded from study, although major depression was not excluded because of its frequent comorbidity with personality disorders.

Patients were kept off all medications for at least 2 weeks and were instructed to follow a low-monoamine diet for 72 hours before the study. All patients were hospitalized for at least one night with an overnight fast and bed restriction before and during the lumbar puncture. The lumbar puncture was performed between 9:00 and 10:00 a.m.; a 20-gauge spinal needle was inserted through the third and fourth lumbar vertebrae. CSF samples from the 12th to 15th cc aliquot were frozen immediately at -80°C until assayed for HVA and 5-hydroxyindoleacetic acid (5-HIAA) by high-performance liquid chromatography (7). The intra- and interassay coefficients of variation in our laboratory are less than 3% and 5%, respectively, for 5-HIAA and less than 4% and 7%, respectively, for HVA.

Data analysis was performed by using analysis of variance and covariance for the group comparisons and the Pearson product-moment correlation for the relationship between the metabolites and symptoms, with two-tailed tests of significance.

RESULTS

The patients with schizotypal personality disorder had higher mean CSF HVA concentrations than the patients with other personality disorders (figure 1). These

differences were not related to gender, presence (current or past) or absence of major depression, freezer time, seasonality, length of hospitalization before the lumbar puncture, or duration of the neuroleptic-free period. Although age ($r=-0.40$, $df=22$, $p=0.06$) and height ($r=-0.58$, $df=22$, $p=0.004$) correlated with CSF HVA, covarying these confounding factors did not affect the significance of the CSF HVA difference between the patients with schizotypal personality disorder and the patients with other personality disorders. Mean CSF 5-HIAA concentrations were not significantly different between the patients with schizotypal personality disorder and the patients with other personality disorders.

Since the psychotic-like symptoms of schizotypal personality disorder are present not only in patients with schizotypal personality disorder but also in patients with other personality disorders, the relationship of these symptoms to CSF HVA was evaluated in both groups combined ($N=24$). CSF HVA concentrations correlated significantly with the number of psychotic-like schizotypal personality disorder symptoms ($r=0.59$, $df=22$, $p=0.002$). The significance of this relationship remained unchanged even after controlling for age and height. The number of other schizotypal personality disorder symptoms (excluding the psychotic-like symptoms) did not correlate with CSF HVA concentrations ($r=0.11$, $df=22$, $n.s.$).

When the effect of psychotic-like schizotypal personality disorder symptoms on CSF HVA was statistically controlled by analysis of covariance, there was no suggestion of group difference between the patients with schizotypal personality disorder and the patients with other personality disorders ($F=0.08$, $df=1, 21$, $p=0.78$).

DISCUSSION

To our knowledge, this is the first investigation of CSF HVA concentrations in patients with schizotypal personality disorder and patients with other personality disorders. The results suggest that CSF HVA concentrations may be higher in patients with schizotypal personality disorder than in patients with other personality disorders and may correlate with the number of psychotic-like schizotypal personality disorder symptoms. Analysis of covariance suggested that the significant differences in the CSF HVA concentrations between the patients with schizotypal personality disorder and the patients with other personality disorders could be largely attributed to the differences in the extent of psychotic-like schizotypal personality disorder symptoms.

Although a psychiatrically healthy control group was not used in this study, the patients with other personality disorders provide a non-schizophrenia-related comparison group derived from the same cohort of patients with personality disorders. The mean CSF HVA concentrations of the patients with other personality disorders in this study were similar to those of healthy control subjects—35.1 ng/ml—in the study of Ballenger et al. (8).

Recently (9), we found that the plasma HVA concen-

trations of a group of patients with schizotypal personality disorder (including eight of the patients in the current report) were higher than those of normal control subjects and that the plasma HVA concentrations positively correlated with psychotic-like schizotypal personality disorder symptoms, paralleling the results of the current study.

Plasma HVA concentrations have been shown to positively correlate with the severity of psychotic symptoms in chronic schizophrenia as well (4, 10, 11). Although a few studies suggested that higher levels of CSF HVA are associated with psychotic and paranoid symptoms of schizophrenia (3), CSF HVA does not display a consistent pattern of abnormalities in chronic schizophrenia (3, 4). However, the interpretation of CSF HVA data in chronic schizophrenia is complicated by a number of factors such as prolonged neuroleptic exposure, drug washout periods, probenecid treatment, brain atrophy, and effects of severe chronic illness. The study of patients with schizotypal personality disorder provides an opportunity to minimize some of these confounding artifacts.

Our results suggest that central dopamine activity may modulate the psychotic-like symptoms of schizotypal personality disorder. In contrast, the social deficit symptoms of schizotypal personality disorder appear to be associated with attentional/information-processing measures, such as eye movement dysfunction (5, 6). Further investigation of schizotypal personality disorder, a less severe disorder in the schizophrenia spectrum, may help to disentangle the biological abnormalities related to psychosis from those related to the social deficit symptoms of the schizophrenia spectrum. The interaction of these two pathophysiological dimensions may contribute to the phenomenological variation along the schizophrenia spectrum (5).

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Impaired Eye Tracking in Undergraduates With Schizotypal Personality Disorder

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Qualitative ratings of smooth pursuit eye movements were significantly worse for 14 undergraduates with DSM-III-R schizotypal personality disorder than for 18 comparison subjects. The groups did not differ on IQ, indicating that deficits in smooth pursuit eye movements in schizotypal personality disorder are not a function of cognitive deficits.
(Am J Psychiatry 1993; 150:152-154)

Impairment of smooth pursuit eye movements has been proposed as a genetic marker for schizophrenia. Such impairments are found in up to 86% of schizophrenic subjects and are also prevalent in their relatives (1). Siever et al. (2) conducted the first study of smooth pursuit eye movements in patients with schizotypal personality disorder diagnosed according to DSM-III; schizotypal subjects selected from a clinical population were found to have worse eye tracking (measured qualitatively) than did normal subjects. This finding supports the findings of a previous study (3) indicating that undergraduates selected on the basis of poor eye tracking have a significantly higher prevalence of schizotypal personality disorder and a study (4) showing impaired smooth pursuit eye movements in subjects with high scores on the Chapman anhedonia scales. However, Thaker et al. (5) failed to replicate the finding of Siever et al. (2) with a group of unhospitalized schizotypal subjects matched with comparison subjects on the basis of approximated IQ. Thaker et al. hypothesized that the findings of Siever et al. may have been an artifact of the severity of the disorder and concomitant cognitive disturbances in the clinical group; Thaker et al. suggested that impairments in smooth pursuit eye movement do not reflect an underlying genotype but, rather, indicate cognitive and functional impairment in schizotypal subjects. Siever et al. (6) noted that the schizotypal subjects in the study by Thaker et al. were diagnosed according to DSM-III as opposed to DSM-III-R. Still, the question remains whether the findings of Siever et al. were specific to an impaired clinical population of patients seeking treatment. The present study tested this hypothesis

by examining smooth pursuit eye movements in a non-patient group of college undergraduates with schizotypal personality disorder diagnosed according to DSM-III-R criteria.

METHOD

College undergraduates (N=822) enrolled in introductory psychology courses were prescreened with the Schizotypal Personality Questionnaire, an instrument based on the nine DSM-III-R criteria for schizotypal personality disorder that has demonstrated reliability and validity (7). Respondents who scored in the top and bottom 10% were invited to participate in the experiment, which involved measurement of smooth pursuit eye movements, neuropsychological testing, and a clinical interview for schizotypal personality disorder. Of those eligible, 64 subjects (30 from the top 10%) volunteered for the entire battery of tests. Informed consent was obtained for all subjects.

Clinical diagnosis of schizotypal personality disorder was determined by using a section of the Structured Clinical Interview for DSM-III-R Personality Disorders (SCID-II) (8). The interrater reliability (coefficient kappa) for presence of schizotypal personality disorder according to the SCID-II was 0.89. Fourteen subjects, all with Schizotypal Personality Questionnaire scores in the top 10%, received clinical diagnoses of schizotypal personality disorder. Eighteen subjects, all from the bottom 10%, who had no signs or symptoms of schizotypal personality disorder were the comparison subjects. This comparison group was selected so as to increase the power of the comparison by minimizing the possibility that the group contained any false negatives for schizotypal personality disorder. The two groups did not significantly differ with respect to gender composition ($\chi^2=1.52$, $df=1$, $p>0.21$), ethnicity ($\chi^2=4.5$, $df=3$, $p>0.21$), or age ($t=0.20$, adjusted $df=23.7$, $p>0.84$).

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To measure smooth pursuit eye movements, Ag/AgCl electrodes were placed on the outer canthus of each eye; electro-oculogram tracings were recorded on a polygraph. In addition, Ag/AgCl electrodes were placed above and below the supra- and infraorbital of the left eye to record eye blinking, and a forehead electrode served as ground. As in the study by Siever et al. (2), the subjects watched a pendulum 1 m away oscillating at 0.4 Hz with an amplitude of 20°. Two trials of 50 seconds each, with a realerting every 25 seconds, were recorded for each subject. The experimenters who recorded smooth pursuit eye movements were blind to Schizotypal Personality Questionnaire results and diagnostic status.

The tracings of smooth pursuit eye movements were scored qualitatively by two raters, blind to Schizotypal Personality Questionnaire results and diagnostic status, using a 5-point system based on that of Shagass et al. (9); higher numbers represented better tracking. This qualitative rating system is comparable to that used by Siever et al. (2). The eye blink record was used to rule out blinks as intrusions. Previous studies (10) have shown qualitative ratings to be correlated ($r=0.86$) with a quantitative measure, the natural log frequency of the signal-to-noise ratio. Interrater reliability in the present study, as determined by the intraclass correlation, was 0.84. The scores given by the two raters were averaged for analysis in the present study.

The subjects were given the digit span, arithmetic, block design, and digit symbol subsections of the WAIS-R. The mean score for the first two was used as an approximated verbal IQ, the mean for the latter two was used as an approximated performance IQ, and the mean of all four was used as an approximation of full-scale IQ.

RESULTS

The two groups' mean qualitative ratings of smooth pursuit eye movements and mean IQs were compared with two-tailed *t* tests. The schizotypal subjects had a mean eye movement rating of 2.57 ($SD=1.04$), and the comparison subjects had a mean rating of 3.36 ($SD=0.98$). This difference was statistically significant ($t=2.21$, $df=30$, $p=0.04$), indicating worse tracking in the group with schizotypal personality disorder.

There were no significant group differences in any of the WAIS-R subscale scores or in the estimated verbal, performance, and full-scale IQs ($t<0.88$, $df=30$, $p>0.38$ in all cases) or in age (schizotypal: mean=19.0 years, $SD=0.9$; comparison: mean=19.1 years, $SD=2.1$) ($t=0.20$, adjusted $df=23.7$, $p>0.84$).

DISCUSSION

These results demonstrate worse smooth pursuit eye movements in a group of college undergraduates clinically diagnosed as having DSM-III-R schizotypal per-

sonality disorder than in comparison subjects. It should be noted that we used a qualitative rating of smooth pursuit eye movement. Previous studies (10) have shown such measures to be highly correlated ($r=0.86$) with a quantitative measure, the natural log frequency of the signal-to-noise ratio, and interrater reliability in the present study was also high (intraclass correlation=0.84). However, qualitative measures are unable to specify the exact character of the impairment. Given the positive findings of the present study, further studies are needed to determine which eye tracking subsystems (e.g., gain, saccades) are specifically involved.

This study addresses the recent suggestions that abnormalities in smooth pursuit eye movements might be found only in cognitively and functionally impaired schizotypal subjects (5) and that research using DSM-III-R diagnostic criteria for schizotypal personality disorder needs to be done (6). First, the schizotypal subjects in the present study were not functionally impaired, insofar as they were all nonpatient college students. Furthermore, the schizotypal subjects in the present study were not cognitively impaired; they did not differ from the comparison subjects on any of the IQ measures. In addition, although advanced age has been correlated with impairments in smooth pursuit eye movements (1), the subjects in the present study were all under 28 years of age and the schizotypal subjects did not differ significantly in age from the comparison subjects. Also, to our knowledge, ours is the first study to examine impairments in smooth pursuit eye movements in schizotypal subjects diagnosed according to DSM-III-R. Thus, these results support previous findings of a relationship between impairments in smooth pursuit eye movements and schizotypal personality disorder (2-4).

Finally, it should be noted that although the subject group in the present study was not large (32 total subjects), small group size can be expected to produce type II errors and therefore cannot account for the positive findings of the present study.

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Transient Compulsive Foraging Behavior Associated With Crack Cocaine Use

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Compulsive foraging behavior associated with use of crack cocaine involves compulsively searching the environment for possibly misplaced pieces of crack. Of 41 crack cocaine addicts evaluated, 33 (80.5%) reported at least some compulsive foraging associated with use of crack; 21 (51.2%) reported such behavior as always associated with crack use. The mean length of time spent in compulsive foraging was 90 minutes. Cocaine-induced foraging may represent a drug-induced model of a type of compulsive behavior.

(Am J Psychiatry 1993; 150:155-156)

Chronic use of stimulants has been associated with "stereotyped examining, searching, and sorting behaviors," which in the past have been variously referred to as "punding," "hung-up activity," and "obsessive-compulsive tendencies" (1). A recent report (2) described the emergence of "senseless repetitive gestures or tasks" in 15 of 55 cocaine abusers studied; six of the subjects most commonly picked through the pile of a rug while searching the area for drugs. In our crack-cocaine-using population, patients commonly describe a compulsive foraging behavior that many of them refer to as "chasing ghosts" or "geeking." The behavior involves compulsive searching for pieces of crack cocaine that the individual believes might have been accidentally dropped or misplaced by someone in the area where the patient has been smoking crack cocaine. When engaged in this compulsive behavior, the person may repeatedly inspect the floor, carpet, furniture, or path he or she took to get to the place where the drug is used. The searching can also include repeated checking of pockets, clothes, and even shoes and socks. Anything found that resembles rock cocaine is carefully examined; materials typically found are pebbles, candle wax, food crumbs, plaster, bits of drywall, and paint chips.

When crack addicts observe compulsive foraging behavior in others, they usually describe it as bizarre, amusing, and often annoying. Although patients are usually aware that the search will be in vain, they describe varying degrees of inability to resist the urge to search; hence the compulsive quality of the foraging behavior. The compulsive foraging is associated with intense craving for cocaine. The onset of the behavior is

usually associated with the exhaustion of the patient's supply of crack cocaine, but many patients describe periods of foraging between "hits." In this study we examined 41 cocaine-dependent men in an attempt to evaluate formally the nature and frequency of cocaine-induced foraging behavior.

METHOD

Forty-one male inpatients who fulfilled the DSM-III-R criteria for cocaine dependence but no other major functional psychiatric disorder and who gave written informed consent to participate were selected for the study. The subjects could have other substance abuse disorders, but their primary and preferred substance of abuse had to be cocaine. A consensus diagnosis for each patient was made by at least two psychiatrists after an interview with the patient and chart review. No patient met the criteria for obsessive-compulsive disorder outside the context of crack cocaine use. The neurological histories and examinations of the patients were unremarkable.

All patients were administered the Cocaine Experience Questionnaire (3) by two psychiatrists; scoring was done by consensus. To the questionnaire were added questions in the same style that were designed to assess the presence and nature of compulsive foraging behavior (the questions are available on request from the last author). Patients were instructed not to consider as compulsive foraging behavior any activities that would meaningfully help them to acquire more crack cocaine (e.g., going out to purchase more of it, obtaining resources to buy more of it, stealing it from others). Patients were asked to estimate the percentage of co-users that they observed engaged in at least several minutes of compulsive foraging behavior. In addition, the Yale-Brown Obsessive Compulsive Scale (4), as applied to the patients' compulsive foraging, was administered and scored by the consensus of the two examining psychiatrists.

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RESULTS

The patients ranged in age from 22 to 50 years (mean=35.2 years, SD=7). The mean age at first use of cocaine was 23.3 years (SD=5.6), and the mean age at the onset of regular use (defined as weekly use for at least 2 months) was 27.6 years (SD=6.6). The mean estimated amount used per week was 11.7 g (SD=11.8), and the mean binge length was 35.7 hours (SD=41.6), with an average of 2.4 binges (SD=1.9) per week.

Of the 41 patients evaluated, 33 (80.5%) reported at least some cocaine-related foraging behavior; 21 (51.2%) reported that compulsive foraging was *always* associated with crack cocaine use. Eight patients (19.5%) denied any compulsive foraging. The mean length of compulsive foraging behavior was 90.1 minutes (SD=114.4). All subjects reported obsessions about misplaced cocaine and craving for more cocaine associated with compulsive foraging. All of the patients who reported compulsive foraging described the behavior as occurring after their last hit of cocaine and the exhaustion of their crack cocaine supply; 21 (63.6%) of these 33 patients also reported that at least some of the compulsive foraging occurred between hits during an ongoing binge.

The mean Yale-Brown Obsessive Compulsive Scale distress score (0=no distress, 4=extreme distress) associated with the compulsive foraging was 2.0 (SD=1.3). The mean score on ability to resist (0=definitely resists, 4=completely yields) was 2.3 (SD=1.3). Eleven (33%) of the 33 patients who described at least some compulsive foraging reported severe or extreme distress associated with it, and eight (24.2%) of the 33 described completely yielding to the urge to forage compulsively. The behavior started within 5 minutes after the exhaustion of the supply of crack cocaine. Only one patient described similar compulsive foraging associated with past intranasal use of cocaine; all of the other patients described compulsive foraging only in the context of crack cocaine use. Seventeen patients reported being able to lessen their compulsive foraging urges with alcohol, five with opiates (including heroin), six with marijuana, three with "downers," one with phencyclidine, and one with cigarettes.

The mean duration of regular cocaine use before the onset of compulsive foraging behavior was 29.5 months (SD=54.2). The mean percentage of co-users that the patients estimated compulsively foraged was 81.4% (SD=24.9%), which is surprisingly similar to the rate of at least some compulsive foraging found in our patient population (i.e., 80.5%).

DISCUSSION

The compulsive foraging behavior described in this report does not seem to be simply a consequence of intense craving for cocaine. It seems quite like a compulsion and is too complex to be a stereotype. Most patients engaging in this compulsive behavior understand

that they search in vain but to some degree cannot resist the impulse to forage. For all of the patients in this study, the compulsive foraging was transient, lasting at most a few hours, and always in the context of heavy cocaine use. Additionally, the behavior did not involve any other compulsive acts (e.g., hand washing), although two of the patients did describe the onset of serious nail biting in the context of their crack addiction, and one patient described a repeated puffing out of his cheeks associated with heavy crack use.

Many individuals will compulsively forage as well as become involved in other more meaningful activities designed to obtain more cocaine. Patients had different street expressions to describe the compulsive foraging, such as "chasing ghosts," "geeking," or "bugging." However, in the dialect of some patients, the meaning of some of these expressions also included the vigilance behavior associated with the paranoia that is seen in cocaine intoxication (e.g., repeated checking of windows and doors).

Abnormalities of brain serotonergic and dopaminergic neural transmission have been implicated in obsessive-compulsive disorder (5) and cocaine abuse (6). Additionally, some brain imaging studies have reported similar findings in cocaine dependence and obsessive-compulsive disorder, including abnormalities of the orbitofrontal cortex (7, 8). One might speculate that cocaine-related compulsive foraging represents a "pure" form of compulsive behavior associated with dysregulation of ancestral brain systems for repeated checking of the environment for potential resources (and reinforcers) associated with the organism's survival. Hence, cocaine-induced compulsive foraging might represent a drug-induced model of a type of compulsive behavior.

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Book Forum

Nancy C. Andreasen, M.D., Ph.D., Editor

PSYCHIATRY AND THE EUROPEAN COMMUNITY

Evaluation of Comprehensive Care of the Mentally Ill: The Transition From Mental Hospital Care to Extramural Care of the Mentally Ill in European Community Countries, edited by Hugh Freeman and John Henderson. London, Gaskell (Royal College of Psychiatrists) (Washington, D.C., American Psychiatric Press, distributor), 1991, 203 pp., £7.50 or \$30.00 (paper).

That the European Community is interested in mental health is surprising, given its image as a promoter of geographic, economic, monetary, and governmental unity. That its Committee on Health Services Research has decided to concentrate on a "search for models" of comprehensive care for the chronically mentally ill is astonishing.

The European Community set this process in motion in 1985, and representatives from at least 14 countries participate. As I understand it, they started by deciding on a common goal (improving services for the chronically mentally ill as countries moved from institutional to community care), set out to design a study on outcome of various systems and programs, and readily realized that they were dealing with huge differences in data availability, research instruments, and patient evaluation tools. They then convened a conference of experts in what we call mental health services research. This book comprises most of the proceedings.

The chapters present 1) what is currently known regarding research methodology on evaluation of treatment, services, outcome, quality, and cost, 2) what research has already revealed in these areas, 3) what individual research is currently going on, such as the Team for Assessment of Psychiatric Services project in the United Kingdom studying the closing of hospitals, and 4) what future research needs and approaches are. The chapters are written by individuals from different countries and not only address the author's area of interest but also provide country-specific information. Thus they are a mixture of comprehensive literature reviews plus analyses of research results, details of individual research projects, and descriptions of local systems and programs.

These chapters would be of interest to us in the United States for at least four reasons. First, they set our experience into a broader context, revealing many shared problems despite our view that European countries are different from us because of their social services orientation. The shared problems include how to effect the move from hospital to community care, the need for data before policy can be established, the need for asylum, the difficulties of doing research on mental health services because of the enormous number of variables, the dilemma of underutilization of some alternatives (e.g., day hospitals), the difficulty of achieving prevention, and the fragmentation of services.

Second, the very great differences among countries and among regions in countries, despite a common database of research and experience and service delivery tools, provides a naturalistic experiment of great importance.

Third, although there is no country that has the problems licked (despite our tendency to think that things must be better elsewhere), some differences in orientation or delivery may be adaptable (e.g., the French *secteur* is different from the American "catchment," the U.K. "core team" is different from the U.S. "multidisciplinary team").

Finally, like so much else in psychiatry, this book demonstrates that if we keep searching for the big cure rather than looking at subgroup responses—responses to medication or type of community care—we are missing an opportunity. The book also reveals that every country has problems as well as partial solutions, but they are different, and examining those differences may result in incrementally improved care for the chronically mentally ill.

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PSYCHOANALYSIS

The Search for the Self, vols. 3 and 4: Selected Writings of Heinz Kohut: 1978–1981, edited by Paul H. Ornstein, M.D. Madison, Conn., International Universities Press, 1990, 844 pp., \$105.00.

Heinz Kohut is one of the few psychoanalysts who had a major, although indirect, influence on DSM-III and DSM-III-R. Narcissistic personality disorder did not appear in DSM-II, and it is in great measure because of Kohut's contributions and their heuristic influence that after the publication of DSM-II a plethora of publications on narcissism appeared in both scholarly publications and the lay press. Paul Ornstein has served us well by editing a selection of Kohut's published writings and letters while Kohut was alive (1). He serves us once again by making available later scholarly work and letters, both published and previously unpublished, from 1978 until Kohut's death in 1981.

Self psychology, not narcissism, was the central interest of Kohut's last years, and Ornstein orients the reader with a carefully considered introductory chapter entitled "The Unfolding and Completion of Heinz Kohut's Paradigm of Psychoanalysis." Ornstein picks up his "guided tour" where he left off in his previous collection, with Kohut having moved to a position where the self was a supraordinate, bipolar configuration at the center of the psychological universe. This tour, like the last one, is highly recommended as one of the clearest and most comprehensive secondary sources on Kohut's work available. Ornstein describes the development and elaboration of Kohut's ideas, providing a detailed four-page diagram showing six Janus-like nodal points and four periods of development of Kohut's thought. It is the fourth period that these two volumes concern.

Kohut had found drive psychology and ego psychology inadequate to explain many important clinical problems. Alternatively, with a constant emphasis on empathy, he established

a psychology that deals with the formation, functions, breakup, and reintegration of the self. Ornstein highlights Kohut's rejection of Freud's stance as a nineteenth-century scientifically objective observer in favor of the stance of an empathic/introspective data gatherer, a perspective that forced Kohut onto new theoretical ground. Certainly, psychoanalysts and dynamic therapists today are far more inclined to use their own empathy, countertransference, and participation in enactments as sources of data than they would have been if Kohut's ideas had not been heard.

Ornstein's introduction subtly takes on criticisms of Kohut's work (without citing the sources) when he denies the claims that Kohut believed self psychologists are more empathic, use a different kind of empathy, underestimate empathy's capacity to err, are unaware of object relations theories, and substitute a new reification (the bipolar self) for the previously reified id, ego, and superego.

Ornstein organizes his "tour" thorough Kohut's last work by focusing on three main areas: empathy, the nature of Kohut's new concepts and theories, and illustrations of refinements regarding the clinical process and cure. As a close and distinguished colleague, Ornstein had the opportunity to question, debate, and discuss these areas with Kohut, and Ornstein's summary reflects this enrichment.

Turning to Kohut's writings themselves, the new and, particularly, the previously published papers add to Kohut's contributions significantly. Some of the most provocative ideas are that the Oedipus complex with its murderous wishes is not a normal developmental landmark but a pathological one; that to Kohut the observer and observed form an unbreakable unit held together by empathy; and that despite the risk of reductionism, self psychology could provide its own typology of personality patterns based on transference data.

Anyone who has not read "The Two Analyses of Mr Z" before will find it to be a remarkable account. Kohut conducted a classical analysis and then, after a gap of several years, a self psychological analysis on the same patient. In my opinion, it is the clearest presentation of how Kohut attempted to change psychoanalytic practice. Another highlight, published in these volumes for the first time, is Kohut's last public address, made at the Fifth Conference of Self Psychology in Berkeley, Calif., a few days before his death. There are also 160 pages of letters that are largely of interest only to the student of self psychology. Even in his private communications, however, Kohut makes clear that he parted from classical theory with reluctance.

An extensive index to all of Kohut's writings completes the fourth volume. Kohut's prose can be difficult and his style at times almost ex cathedra, but the importance of his subject matter and contributions justifies the tough going.

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Theories of Object Relations: Bridges to Self Psychology, by Howard A. Bacal and Kenneth M. Newman. New York, Columbia University Press, 1990, 289 pp., \$37.50.

This book addresses the continuing dialogue between self psychology and object relations theories as to which theory is

most useful for psychoanalytic psychotherapy of the personality disorders.

The book itself has been thoroughly researched and is well organized. It must have been a Herculean task to reduce these complex ideas to their essence and then to describe them in such clear, plain English. The authors provide more or less faithful histories of the major theorists, including Melanie Klein, W.R.D. Fairbairn, Harry Guntrip, D.W. Winnicott, Margaret Mahler, John Bowlby, and Otto Kernberg. These histories are direct, honest, objective, and meticulous in their distinctions, and thus the book can serve as a resource in this field. For each theorist the authors describe the theory, how it compares and contrasts with the others, and its implications for therapy. These long overdue comparisons make self psychology more comprehensible to object relations theorists. The authors conclude each chapter with a comparison illustrating the virtues of self psychology. They recommend that those who are not familiar with the subject read the last chapter—a review of self psychology—first.

It seems to me that a more appropriate title for this book would be "A Self Psychology Reinterpretation of Object Relations Theory." Although it masquerades as an effort to bridge the gap between the two, the book's real purpose seems to be to point out that self psychology not only is an object relations theory but also is superior to all the other theories. In this endeavor the authors often have to tailor reality to their purposes.

Despite the authors' mostly successful efforts to maintain an objective stance, their underlying bias in favor of self psychology breaks through. For example, in reviewing Mahler's work they decry her continued reliance on drive theory and minimize the emphasis she placed on the role of the mother "when we make explicit what is implicit in Mahler's theory of the centrality of the mother's role" (p. 115). No one could have been more explicit about this issue than Mahler, and no one knows this better than I do, since I used this idea extensively in my own theorizing (1).

The authors' bias shows up again when they compare Mahler's pioneer objective child observation research unfavorably to a clinical article by Tolpin (2). As good as Tolpin's article is, it takes some bias to equate scientifically theoretical speculations with objective clinical research.

A key concept in this dialogue is how important for the development of the sense of self or the personality are the drives and structural theory and how important is the role of the mother. Fairbairn, Guntrip, and Winnicott shifted from emphasis on the former to emphasis on the latter, so the mother's profound influence on the development of the self or the personality was in the literature long before Kohut. The authors wonder why these publications did not influence Kohut or why he denied their influence (p. 227). One self psychologist suggested that Kohut may have been one of those creative people who "misread and diminished the work of significant predecessors so as to avoid an anxiety of influence that would paralyze his own creativity" (p. 205).

An equally tenable idea is that to have acknowledged his predecessors would have placed Kohut in the mainstream with other object relations theorists, thereby minimizing his uniqueness, which he may have experienced as a devaluation or narcissistic injury.

The authors note that with the exception of Guntrip, none of the object relation theorists have conceptualized the subject "whose central need is to relate to the object"—i.e., the self. Are they repeating Kohut's need to deny the contributions of others? I published a book on this very subject 8 years ago (3).

The self psychologist does not distinguish between the in-

ternal and the external object, which leads the therapist, in my view, to focus almost exclusively on the patient's internal experience to the exclusion of reality. "In objecting to the concept of a therapeutic alliance based on reality, self psychologists object to the traditional tenet that confers on the analyst the ability to determine objective reality" (p. 268).

Self psychology sees the therapeutic alliance as "the analyst's commitment to comprehend the meaning of the patient's expressions from a perspective within rather than without the patient's subjective frame of reference." To me, this is a contradiction in terms. By joining the patient's narcissistic fusion fantasies the analyst is trying to create a relationship that can be established only by helping the patient to emerge from these defensive fantasies.

This central confusion about boundaries and reality limits pervades self psychology and is further illustrated by its difficulties with diagnosis, with the "selfobject transference" and with the concept of how psychotherapy promotes change and growth.

I have developed a concept of the narcissistic personality disorder within the framework of object relations theory (3). Central to this conception is the distinction between the grandiose projections that are fantasies and the reality of the therapeutic alliance. It is the resolution of the tension between these two, along with the support for the real self found in the therapeutic alliance, that leads to change and growth.

The self psychologist who deemphasizes the real object and the real therapeutic alliance to focus mainly on the patient's self-object transference runs the risk of joining the patient's narcissistic defenses rather than helping the patient to overcome them. As if the treatment were a kind of fantasy reparation—i.e., doing for the patient now what the parents failed to do in the past. The self psychology view of providing self-object functions for the patient comes very close to this idea, but these attempts at a form of reparation reinforce the patient's projections rather than help the patient to emerge from them. These key differences have been further illustrated in another publication (4).

Theories of Object Relations is an important contribution to the dialogue between self psychology and objects relations theory, and I recommend it highly to all who are interested in the field of personality disorders and in the development of ideas.

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Repression and Dissociation: Implications for Personality Theory, Psychopathology, and Health, edited by Jerome L. Singer. Chicago, University of Chicago Press, 1990, 496 pp., \$34.95.

The reader should not be alarmed if a vague sense of trepi-

dation develops on broaching this book. After all, not only is the subject ineffable by its very nature, but the scope of the work is daunting—18 diverse chapters, all brimming with information.

This should come as no surprise. The book is the distillate of a 5-day conference entitled "Repression, Dissociation, and the Warding Off of Conflictual Cognitive Contents." It was held in 1986 at Yale University and was organized by Dr. Singer, the editor of the volume. The contributors are all acknowledged authorities and distinguished investigators in the field of repression, and here they have summarized their work in chapters of a few dozen pages each. Fortunately, Dr. Singer presents the scope and design of the book clearly in the preface, and the chapters deliver faithfully.

The book's two stated goals are 1) to examine repression from a theoretical and empirical point of view and 2) to examine data on repressor personality styles. To do this, repression first is discussed from historical, psychoanalytic, empirical, and nihilistic perspectives. Next, several chapters discuss the role of repression in hypnosis and the phenomenon of dissociation. Finally, a chapter on the cognitive psychology of repression completes the exploration of the "repressive process." The discussion of repressor personality styles includes some theory, analyses of data from psychological testing and longitudinal studies, definitions of new personality types, a new theory of psychological development, and the psychobiology of repression; it concludes with an overview. Whew.

I think that two chapters are outstanding: one on a topic familiar to most psychiatrists and the other on a less familiar topic. Dr. Marshall Edelson presents an exquisite summary of the concept of defense in psychoanalytic theory, including the most concise and clearest exposition of the basic tenets of psychoanalysis I have read, and Dr. Gordon Bower introduces the reader to cognitive psychology, again in a very clear, rigorous way. Both chapters are wonderful introductions/overviews of their fields. In addition, "Repression, Dissociation and Hypnosis," by Kihlstrom and Hoyt, is exemplary in its application of the scientific method to an area where objective measures are difficult to define.

It is unlikely that most readers will read this book cover to cover because there is too much information, but they will pick and choose chapters of personal interest. It is a rich reference, replete with summaries and citations, covering a variety of topics related to the psychology of repression and dissociation. On the other hand, readers who pick up this book because their primary interest is in dissociative disorders are likely to be disappointed; no author discusses this topic directly, and "dissociative disorders" is not even listed in the index. The mid-1980s, when the conference was held and the chapters were written, was just the beginning of the current wave of research into the dissociative disorders. Clearly the full significance of those (then) recent discoveries was unappreciated.

The problem is, even for a multiauthored book, four years is too long between conference and publication. A thorough, scholarly discussion of repression and dissociation is timely, but a book published in 1990 needs to include the discoveries of the previous half decade. Thus, the reader interested in dissociative disorders would do better to look elsewhere for information. However, readers seeking a thoughtful, detailed, eclectic discussion of the scientific and theoretical basis of repression and dissociation will find much to pique their interest here.

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CHILD PSYCHIATRY

Difficult Moments in Child Psychotherapy, by Stewart Gabel, M.D., Gerald Oster, Ph.D., and Cynthia R. Pfeiffer, M.D. New York, Plenum Medical, 1988, 207 pp., \$37.50.

Most psychiatrists who have worked with children, even briefly, have a story about an awkward or difficult moment with a child: a child who will not come to the therapy room, a child who runs out of the room, abruptly ending the session. These situations rarely are discussed in supervision before they happen; they are usually examined only after the fact. In this brief but excellent book, the authors help child psychotherapists anticipate these situations, not as potential disasters but as problems with a number of possible solutions and as opportunities to gain insight into the child's problems.

The first and third chapters, which take up most of the book, are devoted to examples of difficult experiences, some real and some fictional. The first chapter contains well-written, sometimes humorous vignettes, with characters such as Stubborn Smith, Dr. Angry Redface, and Dr. Eager Learner, depicting many of the difficulties that can arise in working with children and their parents. These situations include the child who tells you that he or she is suicidal or has been abused, the child who is aggressive or sexually provocative, and many more. Following each vignette is a thorough discussion of practical ways the therapist can manage these difficult moments along with the implications of each choice. The authors emphasize that there are no "right" answers.

The third chapter contains anecdotes depicting difficult situations, many felt to be pivotal moments in therapy, from the careers of 11 nationally known child psychiatrists, including Jules Bemporad, John Schowalter, John McDermott, and Clarice Kestenbaum, among others. These stories and the reflections of their authors are particularly useful for demonstrating how the therapist can make use of difficult situations to move the therapy forward. By illustrating a number of different situations that may arise in clinical practice, chapters 1 and 3 allow the inexperienced clinician to think about how she or he might respond to these situations for the benefit of the child.

The second chapter draws on the child psychotherapy literature (with numerous references) to provide a theoretical framework for thinking about difficulties in therapy. The authors believe that many of the expectations that therapists have concerning the process and meaning of psychotherapy are not shared by the parents and the children. When these expectations are in conflict, difficult moments may arise. For example, most therapists believe that unwanted feelings or behaviors in children are caused by psychological problems or conflicts. Parents and children, however, may have different assumptions about the child's behavior, including the belief that the child is "bad," and may not recognize that behaviors can be motivated by feelings that are unknown to the child. Resistance to therapy by the child or termination of therapy by the parents may occur if the parent-child relationship or the family's equilibrium is disturbed when their beliefs are challenged.

The concepts in this brief chapter are illustrated with examples that are not as colorful as the vignettes used in the other chapters. These concepts would have been more easily understood and better illustrated if they had been integrated into the examples of the first and third chapters instead of being placed in a chapter so easily overshadowed by the entertaining vignettes in the rest of the book. Despite this difficulty, I be-

lieve that this chapter is useful to the therapist who has lived through the difficult moment and would like to reflect on how the parent's or child's behavior relates to the particular psychological issues of the family.

In summary, this book examines difficult moments in child psychotherapy from two perspectives, anecdotal and theoretical, providing inexperienced clinicians with starting strategies and a theoretical framework for dealing with these situations. I enthusiastically recommend this entertaining but theoretically solid book to clinicians who treat children, particularly trainees, to help quell the anxiety of learning to work with children and to enable them to turn difficult moments into productive events in therapy.

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Brain and Behavior in Child Psychiatry, edited by A. Rothenberger. New York, Springer-Verlag, 1990, 375 pp., \$109.00.

Perhaps you can judge a book by its cover. This book has on its cover a charming picture of the brain and its functions made by the children of the editor. Not only is this depiction wonderfully childlike and charming, its inclusion indicates that the editor understands and cares for children.

This volume brings to the general reader the contributions of 41 distinguished investigators who share the common goal of understanding and treating children with psychiatric problems, learning disorders, and behavior difficulties. Many different methods of investigating the biological basis of psychiatric disorders are presented along with inferences that can be made regarding normal and abnormal brain function.

Child psychiatry is intrinsically more difficult than adult psychiatry, not only because of the quite variable rates of development between individual children but also because of the different rates of development of different functions within the same child. Nowhere is the nature/nurture interplay more obvious or more complicated than in the case of the developing child, with the complex input of genetic, biological, and environmental factors.

Research in child psychiatry is also quite challenging because of ethical considerations that limit the use of invasive studies and make it difficult to conduct double-blind controlled drug studies. Additionally, the fact that most behavior problems of children are not handled by psychiatrists makes it difficult to generalize from psychiatric settings. Another problem is that the whole educational establishment seems to be based on the proposition that children are blank slates on which experience writes, resulting in a very biased sample of children who are referred for treatment.

This valuable volume has brought to English a great body of work from European child psychiatry along with some American work representing the state of the art in the many different approaches to the fundamental question of brain research.

Freud emphasized the importance of early childhood experience but lacked the tools available to us today. This book has the advantage of neurophysiological studies that include EEG, magnetic resonance spectroscopy, brain mapping, and single photon emission computed tomography, and we also have the powerful tools of epidemiology and molecular genetics.

Brain and Behavior in Child Psychiatry is organized into seven areas. The first section, General Aspects, examines the philosophy, psychobiology, neurophysiology and neurology, biochemistry, and genetics of child development and child psychiatry. This occupies the first 100 pages of the book and

provides an in-depth view of the basic science required for further study. The next section, Cognition, includes chapters on specific developmental learning disabilities and dyslexia. The third section, Speech and Language, has an excellent summary of stuttering and the other varieties of speech disorder. The fourth section, Childhood Psychoses, examines autism and schizophrenia. The fifth section, Minimal Brain Dysfunction and Head Injury, considers the implications of these conditions for the later development of psychopathology. The sixth section, Sleep, is an excellent summary of a very difficult and complicated area. It is practical and lucid and should be read by every pediatrician as well as every psychiatrist dealing with children. The final section, Diagnostic and Therapeutic Aspects, discusses the use of psychotropic drugs, biofeedback, and the important topic of developing psychophysiological indexes. More than just a cookbook, this section gives a method and a direction for future research.

Brain and Behavior in Child Psychiatry is a useful volume that succeeds in a way most multiauthored books do not in integrating an area of fundamental importance to the clinical discipline of child psychiatry. The book should be read by all psychiatrists, not just those who specialize in children, because of the developmental basis of adult disease and the important insights to be gained into genetics, brain damage, and the other factors that are often overlooked when treating adults. Some of the ways of examining the topic that might be called behavioral neurology or neuropsychiatry also have general application to other populations. Even if one is more interested in the clinical than the research aspects of behavior, this book remains an interesting and valuable reference.

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Textbook of Child and Adolescent Psychiatry, edited by Jerry M. Wiener, M.D. Washington, D.C., American Psychiatric Press, 1991, 636 pp., \$95.00.

Two major textbooks on child and adolescent psychiatry were published in 1991: the book under review, edited by Jerry Wiener, and *Child and Adolescent Psychiatry: A Comprehensive Textbook*, edited by Melvin Lewis (1). In this rapidly expanding field, both references are valuable additions to the existing literature. Whereas the text edited by Lewis reviews the scientific basis and clinical aspects of child and adolescent psychiatry, the major goal of Wiener's text, as noted in the book's introduction, is to provide a "clinically focused" textbook. This book is divided into five sections. The first, The Field of Child and Adolescent Psychiatry, begins with "The History of Classification in Child and Adolescent Psychiatry" and is a prelude to the emphasis of this book on a review of DSM-III-R disorders. The next section includes 12 chapters on assessment and issues in diagnosis. The chapter entitled "Concepts of Diagnostic Classification," a review of substantive conceptual issues in classification is one of the more informative chapters in this section. Unfortunately, several chapters in this section are quite short and are likely to be useful to clinicians in training only as sources for locating other, more detailed reviews on assessment. Also, the chapter entitled "Psychological and Neuropsychological Testing" focuses heavily on the assessment of personality and psychopathology and does not adequately discuss the assessment of academic abilities.

The majority of the text, sections three through ten, is dedi-

cated to a review of DSM-III-R childhood and adolescent psychiatric disorders. This is one of the strengths of this text. These are thorough and easy-to-read reviews that cover basic aspects of the major psychiatric disorders affecting this age group. The chapters on autism, anxiety disorders, anorexia nervosa, tic and movement disorders, and substance abuse are particularly informative and well written. Section ten, Special Issues in Childhood and Adolescence, includes reviews of several psychiatric disorders and has several chapters on selected issues in the field such as child abuse, suicide, and personality disorders. Although brief, the chapter entitled "Suicide and Suicidality" provides a very good data-based review of this topic. Readers will need to refer to other sources to review additional important issues not included in this section, such as the effects of divorce, adoption, fostering, and institutional rearing, and cross-cultural issues in child and adolescent psychiatry.

The final section in this book reviews a variety of treatment modalities. This section is broadly based and includes good review chapters on psychopharmacology, psychodynamic psychotherapy, short-term psychotherapy and crisis intervention, family therapy, behavioral therapy, group therapy, and hypnosis.

The strength of this text is its thorough and easy-to-read review of DSM-III-R psychiatric disorders and their treatment. The reader may be somewhat frustrated by the lack of a chapter synthesizing current knowledge about the epidemiology, etiology, and possible mechanisms underlying the development of childhood and adolescent psychiatric disorders as well as their continuities and discontinuities with adult psychopathology. Overall, this text is an excellent resource. Beginning students in child and adolescent psychiatry will find the sections on assessment and psychotherapeutic treatments to be worthwhile introductions to these areas. Both beginning students as well as more experienced clinicians will find the sections that review psychiatric disorders, special issues, and pharmacological treatment to be useful references for clinical practice.

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The Clinical Interview of the Child, 2nd ed., by Stanley I. Greenspan, M.D., with Nancy Thorndike Greenspan. Washington, D.C., American Psychiatric Press, 1991, 238 pp., \$36.50.

As child psychiatry residents and fellows go through their training, they quickly realize that children, especially young children, are not merely small adults and that the interview techniques used with adults are often frustrating if not downright counterproductive when applied to children. One of the great hurdles of training is to develop a systematic, sensitive way of observing and interviewing children; one of the struggles for instructors and supervisors is communicating their own system to their trainees. In this book, a master child clinician accomplishes this second task very well, helping the reader to accomplish the first one.

Readers unfamiliar with Dr. Greenspan's developmental

theories will need to carefully read the first chapter, "Conceptual Foundations: An Overview," to understand the framework and language of the subsequent chapters. (This developmental theory is more thoroughly explained in the author's recent work, *Infancy and Early Childhood* [1] as well as his other works.) In this "developmental structuralist" approach, the course of early childhood development is divided into "four core processes, or levels. They involve how a child 1) attends and engages, 2) communicates with gestures and behaviors, 3) creates internal mental images (ideas) and shares them with others (i.e., symbols, mental representations), and 4) categorizes these meanings and makes connections between them" (p. 6). In assessing a child, the first step is to determine to what extent each of these core processes has been attained. Second, the child is observed for the range and richness of themes communicated at each level. Third, the clinician attempts to determine the stability of each level (e.g., in times of emotional stress or anxiety, does the child regress from well-elaborated verbal communication to gestures and behaviors?). The child may express different themes at different levels; for example, she or he may have achieved representational elaboration and differentiation in the communication of dependency but still rely on gestures and behaviors to express anger or aggression.

This model is used to provide a consistent framework for the remainder of the book, which is largely composed of clinical vignettes and discussion based on these vignettes. The clinical cases involve children of all ages and present only the interview with the child (or the child and parent together in the case of very young children) rather than an extensive case history. By using this technique, Dr. Greenspan forces the reader to focus in on what can be learned from the child. At the end of each clinical illustration, Dr. Greenspan records his own subjective reactions to the encounter, along with summary comments interpreting the contents of the interview according to his developmental framework. A later chapter provides more complete formulations of the cases based on the same model.

It becomes evident as the book progresses that the author is not an ardent fan of DSM-III-R diagnoses. (Many in child psychiatry see glaring shortcomings in the present system.) In the chapter entitled "Constructing a Formulation" he presents his own nosology based on the developmental structuralist approach. This presents categories in terms of characterological constrictions and encapsulated symptoms or character disorders. A table is provided to relate these diagnostic categories to the operationally defined DSM-III-R categories. In reviewing this approach, it becomes clear that a child might be described accurately by one of the developmental categories but still not meet criteria for a DSM-III-R diagnosis. Developing a diagnostic system that is clinically useful, reliable, and valid is an ongoing issue in all of psychiatry, and it will be interesting to see if some of these developmentally based schemes are incorporated into future editions of DSM.

This new edition of *The Clinical Interview of the Child* is an excellent resource for both the novice and the experienced clinician. The style is relaxed, almost conversational, and therefore a pleasure to read. Dr. Greenspan's approach to observing children makes sense, and that many readers will finish this book, close the cover, and sit back smiling and thinking, "Hey, I can do that, too," attests to his success in presenting it clearly.

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NORMAL DEVELOPMENT

New Dimensions in Adult Development, edited by Robert A. Nemirow, M.D., and Calvin A. Colarusso, M.D. New York, Basic Books, 1990, 531 pp., \$39.95.

Life changes, determined by mysterious, transcendent, constant, and imminent influences, are inevitable. We struggle through many metamorphoses to acquire a permanent notion of ourselves. Like adult development itself, this book proceeds in fits and starts, with delightful interludes and thought-provoking confrontations.

The book's 22 chapters are written by 27 experts in such various disciplines as psychoanalysis, child development, psychology, anthropology, and sociology. A discussion by the editors follows each chapter. The entire book is divided into four parts. Part one, Transition to Adulthood, captures the integration of the oedipal and pre-oedipal themes of earlier developmental stages with the societal demands and the biological requirements of late adolescence, necessary to the erection of competent ego structure. *New Concepts in Adult Development*, part two, is the main course of the book. Here the editors develop their theory of adult development based on fulfillment of dependency wishes and the recognition and acceptance of personal death. This theory is compared with theories of cognitive, motivational, affective, behavioral, and gender-oriented development to achieve a better understanding of the ongoing process of maturation. *Clinical Perspectives*, part three, attempts to establish the usefulness of developmental processes in approaching age-specific issues such as the awareness of one's own near death or the catastrophic reaction to a loss. These processes also help to understand the ever-changing quality of the ego throughout the life cycle both as an individual and as a couple. Finally, *Applications of Adult Developmental Theory*, part four, addresses the common effect that unthwarted developmental processes share, regardless of personal adult past experiences, culture, and gender. This effect has the "feel" of wisdom defined as a greater propensity for empathy and tolerance, an exquisite sensitivity regarding a significant other's needs, an enduring capacity for altruism, and an enhanced ability to reach mutually satisfying solutions to personal problems.

New Dimensions in Adult Development is a guidepost to the understanding of "normal," delayed, and abnormal developmental processes encountered in the clinical field. It promotes an open-minded attitude toward the different ways we live our lives. The core message of this impressive work is the ongoing "restorative work" on the ego that remains constant throughout the life cycle. I particularly enjoyed Dr. Gutman's "stressful passage toward androgyny" when parents experience a change in their respective gender-oriented roles as their children become physically and psychologically independent. Each parent then assumes characteristics of the opposite sex and engages in a potentially destructive or potentially fulfilling situation.

Some reproaches are in order. As in any multiauthored publication, the book lacks literary unity, and certain aspects of adult developmental stages, such as the reparative work on an

aging body, would apply only to middle-class college graduates in Western culture.

I strongly recommend this book to all the disciplines involved in improving the human condition or dedicated to understanding the many facets of human nature.

ALEXANDRE CARRE, M.D.
New Haven, Conn.

TEXTBOOKS

The Clinical Interview Using DSM-III-R, by Ekkehard Othmer, M.D., Ph.D., and Sieglinde C. Othmer, Ph.D. Washington, D.C., American Psychiatric Press, 1989, 485 pp., \$38.50.

Conducting a clinical psychiatric interview is an art by itself; it entails making patients feel at ease so they can talk freely about extremely personal issues. I think this is learned by observing and being observed while conducting an interview followed by a critique and would be difficult to acquire solely from a book. In this book, however, the authors have skillfully organized the various aspects into nine chapters that present essential information for a successful diagnostic interview.

The book begins by describing insight- and symptom-oriented interviewing and their individual goals, with a good account of how the two can complement each other in a two-step approach. Developing rapport with the patient is very important, and this is excellently described with the help of case vignettes. The examples illustrate how transitions can be made from one topic to another during an interview when a lot of territory needs to be covered in a limited span of time. In addition, various strategies of obtaining information, such as probing, echoing, and summarizing, are discussed. I was especially impressed by the section on mental status assessment, which I thought was extremely useful, along with the chapter "Seven Steps to Make a Diagnosis," written from the point of view of the oral psychiatric boards. Using two interviews as examples, the authors show how the various strategies can be applied in the case of a cooperative patient and in the case of an uncooperative patient. The book conveys very clearly how the process of inclusion and exclusion of diagnoses should occur during the interview process. Personality disorders, which are usually difficult to treat, complicate the axis I diagnoses, and are often a diagnostic dilemma, are thoroughly discussed in terms of diagnostic strategies and specific approaches to the individual disorders. Disorders of thought, such as circumstantiality and tangentiality, are clearly illustrated along with a detailed glossary for beginners in the field.

Some parts of the book might be misleading for beginners, such as the table indicating correlations of major psychiatric disorders to personality disorders. For example, schizophrenia, undifferentiated type, with negative symptoms is reported to correlate with schizoid personality disorder, and obsessive-compulsive disorder is shown to be correlated with obsessive-compulsive personality disorder. This might be true to some extent in terms of symptoms but not necessarily in terms of the disorder.

Technically, the book is of high quality with good editing. Typographical errors and misspellings are rare. At various points the authors use the words "client" and "patient" interchangeably, but the writing style overall is good. This book would certainly prove very useful for beginners in the field, especially psychiatry residents, because it would complement the interviewing course offered by the training program. This

book would be useful for all mental health professionals because it serves to enhance clinical skills.

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Psychiatric Case Formulations, by Len Sperry, M.D., Ph.D., Jon E. Gudeman, M.D., Barry Blackwell, M.D., and Larry R. Faulkner, M.D. Washington, D.C., American Psychiatric Press, 1992, 167 pp., \$20.25.

The formulation of psychiatric cases is a formidable task. The authors, citing Larry Faulkner et al. (1), define case formulation as "a prescribed method for the orderly combinations or arrangement of data and treatment recommendations about a psychiatric patient according to some rational principles" (p. 1). As the authors imply, case formulation is at the core of clinical psychiatry and entails all aspects of psychiatric care. In short, it is the process by which we understand patients and their illnesses and decide how best to treat them. Thus, a book on psychiatric case formulations involves the entire field of psychiatry.

The authors of this book appear well-attuned to the complexities of their task and organize their book in a cogent and concise manner. The book is divided into eight chapters. The first is devoted exclusively to an in-depth and complete discussion of philosophical and theoretical concerns and includes an appendix. The next four chapters cover the major paradigms: psychodynamic, biological, behavioral/cognitive, and biopsychosocial. Each of these chapters begins with a discussion of the theoretical basis of its paradigm and ends with the formulation of a case according to its theoretical precepts. The authors, using an interesting pedagogic technique, present the commonly encountered case of a patient with major depression, obsessive-compulsive personality disorder, and social issues in the first chapter. In the subsequent chapters they formulate the same case using the four different paradigms. In chapter six the authors present their view on the convergence of these paradigms and their assertion that an eclectic, biopsychosocial formulation is the most appropriate and useful for psychiatric practice. The final two chapters present practical methods for constructing a formulation by providing written examples.

The authors strive to be comprehensive in both their theoretical and their clinical discussions. They examine terms that have been overused and trivialized (such as "biopsychosocial") and explain their meaning and purpose in a scholarly manner. Their use of the same case to illustrate the various paradigms is both clever and appealing. This allows the authors to make a strong argument for a convergence of paradigms and the necessity of the biopsychosocial perspective.

The authors' concern for completeness and inclusiveness may be both a blessing and a curse. Their inclusion of vast quantities of both theoretical and practical information in a few pages sometimes leads to a very dense text that detracts from its stated desire to teach the skill of case formulation. It might have been better either to have written a longer book or narrowed their scope to their preferred biopsychosocial paradigm.

Psychiatric Case Formulations is a well-researched book written by psychiatrists who demonstrate both scholarship and clinical acumen. The last two chapters, which focus on the nuts and bolts of writing a case formulation, are excellent and would be of great value to the beginning clinician or anyone honing a presentation to managed care or other reviewers.

The earlier chapters would be of more use to those who are knowledgeable about and are interested in building psychiatric models and paradigms. This book would be of particular use to those who are involved in psychiatric education and residency training.

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STEVEN BERKOWITZ, M.D.
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Handbook of Affective Disorders, 2nd ed., by E.S. Paykel. New York, Guilford Press, 1992, 699 pp., \$65.00.

The first edition of this handbook was published in 1982. This second edition, published a decade later, reflects updated information and research related to mood disorders.

The book has five major sections. The first focuses on descriptive aspects, including symptoms of depression and mania, concepts in classification, history of mood disorders, the bipolar/unipolar distinction, the relation of anxiety to depression, and course and outcome of affective disorder.

Part two consists of 12 chapters related to causative aspects. These chapters cover epidemiology, genetics, life events, early environment, personality, psychodynamics, animal models, neurochemistry, neuropsychology, and sleep.

Part three consists of eight chapters on medication and physical treatments, including tricyclics and monoamine oxidase inhibitors, ECT, lithium, anticonvulsants, and maintenance treatment. In addition, there are chapters on resistant depression and psychosurgery as well as prediction of treatment response.

Part four consists of six chapters on psychotherapeutic, cognitive, and social treatments, including psychoanalytically oriented psychotherapy, group therapy, family/marital therapy, interpersonal psychotherapy, cognitive therapy, and social community approaches.

The last section consists of nine chapters on special aspects, including transcultural topics, seasonal affective disorder, postpartum depression, depression in children and adolescents, depression in old age, bereavement, suicide, and depression in general practice and other medical settings.

Thus, the *Handbook of Affective Disorders* is a comprehensive presentation of past research and the current status of mood disorders. The book is multiauthored, and each of the contributors seems to have been carefully chosen to represent a high degree of expertise in his or her field. The references go through 1991, and the index is quite useful for topic references. This is an international group of authors, with contributors from the United Kingdom, the United States, Denmark, Australia, Italy, Germany, New Zealand, and Sweden. The chapters are well referenced; there are approximately 50 and in many cases more references per chapter.

The editor's intent for this volume was to provide a comprehensive handbook that would be "useful to students, postgraduate trainees, clinicians and research workers in psychiatry and related medical, scientific, nursing, psychological, social work and mental health disciplines." I suspect that the book will more than adequately serve the editor's purposes.

Indeed, it is a very useful book to have for a quick update on topics related to mood disorders and a handy reference list. I think it would also be useful to psychiatric residents or others who are about to take their boards in psychiatry because the chapters are clear, well written, and provide comprehensive views of their topics.

Dr. Paykel should be congratulated for assembling this fine group of contributors and for his editing efforts.

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Manter and Gatz's Essentials of Clinical Neuroanatomy and Neurophysiology, 8th ed., by Sid Gilman, M.D., and Sarah Winans Newman, Ph.D. Philadelphia, F.A. Davis Co., 1992, 328 pp., \$21.95 (paper).

The authors of the eighth edition of this textbook have maintained its tradition of explaining physiological concepts within the context of the anatomical organization of the nervous system. This is a short but comprehensive and well-written review of the human nervous system.

The book is divided into seven sections and 30 chapters. It starts with a good account of basic principles. Here the authors briefly but very clearly discuss some of the tricky areas like the development of the nervous system and electrophysiology of the nerve cell. The second section deals with peripheral and autonomic nervous systems with a good emphasis on physiological aspects. Section three, on ascending and descending tracts, is the best in the book. The modern understanding of these tracts is presented clearly and authoritatively with excellent illustrations. Clinical disorders and syndromes are highlighted in appropriate places.

The fourth and fifth sections deal with the brainstem, cerebellum, cranial nerves, and higher levels of the nervous system. The chapters in these sections are divided into functional units in contrast to anatomy books that describe the brain by sections and physiology books that describe by systems. This makes the book easy to understand and interesting to read. It also helps the reader understand the lesions at different sites and their clinical presentation. Cerebral circulation and CSF are discussed in the new sixth section. In this eighth edition the authors have added for the benefit of medical students a section on clinical approaches to the patient with neurological disorder.

Chapters dealing with modern concepts and recent increases in understanding of many areas like the sensory system, neurotransmitters, and higher functions of the brain have excellent discussions and are very informative. This book has plenty of good illustrations and tables. The index is thorough and complete.

Although the book is written for medical students, it will be useful to anyone for a quick review. With the recent advances in neuroimaging research in psychiatry, a thorough knowledge of neuroanatomy and neurophysiology has become essential for a psychiatrist. *Manter and Gatz's Essentials of Clinical Neuroanatomy and Neurophysiology* is the right book for an update of the subject.

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Reprints of Book Forum reviews are not available.

Letters to the Editor

Antidepressant Withdrawal: Prospective Findings

SIR: The literature suggests that the withdrawal of tricyclic antidepressants precipitates syndromes attributable to cholinergic rebound in discrete areas of the brain or autonomic nervous system (1). These syndromes are 1) anorexia, nausea, emesis, diarrhea, diaphoresis, myalgias, malaise, headache, chills, fatigue, and anxiety; 2) insomnia accompanied by vivid dreams; 3) akathisia or parkinsonism; and 4) hypomania or mania. The prevalence of these syndromes was assessed among one outpatient and nine inpatients at the psychiatric clinic of the University of Pisa using a prospective design.

The prewithdrawal evaluation included a physical and neurological examination, laboratory screen, and completion of the 21-item Hamilton Rating Scale for Depression (2) and Present State Examination (PSE) (3). The subjects were then serially evaluated by the same physician following the withdrawal of antidepressants. Symptoms were monitored by spontaneous responses to open-ended questions such as, "How do you feel today?"

One woman (age=52 years) and nine men (mean age=50 years, SD=8.6) consented to participation. Nine subjects met the DSM-III-R criteria for major depression. One met the criteria for schizophrenia. The patients were subjected to the abrupt withdrawal of clomipramine (N=2), amitriptyline (N=2), imipramine (N=1), maprotiline (N=1), and amineptine (N=1), and a combination of maprotiline and amitriptyline (N=1), clomipramine and amineptine (N=1), or clomipramine and amitriptyline (N=1). The patients were not subjected to the withdrawal of other agents.

Seven patients experienced symptoms or exhibited signs previously associated with the withdrawal of antidepressants. Four subjects experienced some combination of malaise, anorexia, nausea, vomiting, headache, or chills and diaphoresis (syndrome 1) following the withdrawal of imipramine (N=1), amitriptyline (N=1), amineptine (N=1), or clomipramine in combination with amitriptyline (N=1). Six individuals developed insomnia (syndrome 2) following the withdrawal of imipramine (N=1), clomipramine (N=2), amitriptyline (N=1), amineptine (N=1), or clomipramine in combination with amineptine (N=1). Two of these subjects had unusually vivid dreams. One subject developed ventricular extrasystolic beats following the withdrawal of amitriptyline. Another individual exhibited mild to moderately severe resting tremor of the jaw, tongue, upper extremities, and rigidity for 4 days following the discontinuation of amineptine. Patients withdrawn from amitriptyline (N=1) and imipramine (N=1) met the criteria set forth in the PSE for hypomania. Neither had a history of hypomania.

The mean baseline Hamilton depression scale score of the subjects was 17.7 (SD=8.1). The mean score 14 days after the discontinuation of the antidepressants was 12.3 (SD=9.6) (n.s.). This is consistent with the results of a retrospective study (4). However, in both instances severity was measured in depressed subjects who were unresponsive to the antidepressant which was discontinued.

The withdrawal of all but one agent (maprotiline, N=1)

precipitated phenomena previously associated with the withdrawal of antidepressants. This patient developed *emuresis nocturna*.

The findings presented are consistent with retrospective reports of the adverse effects of withdrawing antidepressants. The subjects developed each of the four syndromes described in the literature. One patient even developed an arrhythmia upon the discontinuation of amitriptyline. Similar arrhythmias were previously reported to occur upon the withdrawal of two other antidepressants imipramine (1), and clomipramine (5).

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Tardive Dyskinesia-Like Syndromes With Clomipramine

SIR: A number of cases of tardive dyskinesia-like syndromes have been described in association with the use of tricyclic antidepressants (1-5). Clomipramine has been implicated in a few cases as well, which are said to have developed over a period of several weeks to months, frequently in association with haloperidol (personal communication, CIBA-GEIGY). To the best of my knowledge, this syndrome has always resolved with the cessation of clomipramine except in cases where a neuroleptic was also involved. The following case illustrates the development of a severe tardive dyskinesia-like syndrome in a woman who was also taking thiothixene.

Ms. A was a 51-year-old woman who carried a diagnosis of paranoid schizophrenia with delusions and auditory hallucinations. She had experienced akathisia secondary to thioridazine in the past and also had experienced a mild form of orobuccal dyskinesia. At the time she first came to my

attention there was no evidence of abnormal movements and the akathisia was under control. She was taking thiothixene, 10 mg t.i.d., buspirone, 10 mg t.i.d., and trihexyphenidyl, 5 mg b.i.d.

Because of persistent obsessive behavior related to money and cigarette smoking, she was started on a regimen of clomipramine, 50 mg at bedtime. In 1 week, she complained of a restless feeling along with dry mouth. Two days later she rapidly developed involuntary orobuccal movements consisting of severe lip smacking and puffing as well as tongue protrusion. Her speech was slurred and she experienced serious difficulty in swallowing. She manifested repetitive stereotypical movements of her arms, placing an arm behind her head, and moderate choreoathetosis of the arms. Repetitive retracting movements of the abdomen were evident along with a truncal rocking motion. She constantly crossed and uncrossed her legs and tapped her feet and was unable to walk because of lower extremity restlessness and incoordination.

Clomipramine was discontinued, the thiothixene was decreased to 10 mg b.i.d., the buspirone was decreased to 5 mg t.i.d., and clonazepam was begun at 1 mg b.i.d. The next day Ms. A was still unsteady but felt better and displayed far fewer movements. Thiothixene was further decreased to 5 mg t.i.d. and clonazepam and trihexyphenidyl were continued. Four days later buspirone was discontinued and almost no movements were in evidence. Ten weeks later there were no signs of any abnormal movements nor did the patient report any subjective sensation of restlessness or other discomfort.

This case illustrates an exceedingly rapid development and equally rapid remission of a tardive dyskinesia-like syndrome which may have been more severe than those cases previously reported to the manufacturer. The syndrome cannot be entirely attributed to clomipramine, as the patient was taking a neuroleptic and was obviously predisposed to the development of a movement disorder. As with previously reported cases, this syndrome remitted once the clomipramine was discontinued.

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Huntington's Disease and Propranolol

SIR: In addition to movement disorder, Huntington's disease is almost invariably accompanied by a wide variety of psychiatric and behavioral problems (1). Irritability and aggression are especially problematic in this patient population and have been reported to respond favorably to propranolol (2). My

colleagues and I recently treated a patient with Huntington's disease and a different sort of behavioral problem who responded well to propranolol.

Ms. A, a 46-year-old woman with a 2-year history of Huntington's disease, was admitted for medication readjustment. She had complained of insomnia to a local physician and was ultimately placed on a combination of chlorpromazine, pimozide, trazodone, clonazepam, and chloral hydrate. There was no past psychiatric history. On admission Ms. A was extremely akinetic and almost mute. Over the next 7 days medications were withdrawn and within 2 weeks her speech and motor activity were back to baseline. Medication at that time included only lorazepam, 1 mg h.s., resulting in 6-8 hours of uninterrupted sleep per night.

Over the subsequent weeks, the patient's behavior was noted to be characterized by a pervasive sense of intrusiveness, urgency, and "desperation." She would almost continually approach nursing and medical staff, asking for medication and/or reassurance because of her movements (which were in fact rather mild), difficulty sleeping, or the absence of her husband. She would frequently become tearful and follow staff when an attempt was made to end the discussion. No irritability or hostility was noted, and she denied any symptoms of depression.

Approximately 3 weeks after withdrawal of other medications, propranolol was begun and titrated to a final dose of 30 mg q.i.d. Within 1 week, Ms. A no longer exhibited any intrusive or "desperate" behavior. She rarely voiced any complaints when approached and expressed her ability and willingness to "live with" the movement disorder and the temporary separation from her husband during hospitalization. She was ultimately discharged to the care of her husband on a regimen of propranolol and lorazepam.

Although a specific count of the number of times per day that the patient approached staff would have reflected the noted improvement, it would not have captured the dramatic improvement in the elements of desperation and impatience, elements that might have contributed to the referring physician's seemingly desperate pharmacologic regimen.

In our previous work with aggressive patients with Huntington's disease, it was noted that most aggressive episodes were triggered by minor frustrations that would be disregarded by most individuals (2). In these patients, however, an aggressive response to these frustrations is no longer inhibited. In the same way, we postulate that our patient responded to such minor frustrations with fearfulness and desperation, rather than aggression. It is conceivable that the essential mechanism of action of β blockers in such cases is to attenuate the disinhibition that results from damage to frontal and subcortical systems. β Blockers have been studied extensively in the management of aggression in patients with mental retardation, head injury, and a variety of other organic mental disorders (3, 4). This report raises the question of whether there are other aberrant behaviors in these populations, such as intrusiveness, that might also be amenable to treatment with these agents.

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Euphoria With Buspirone After Fluoxetine Treatment

SIR: Buspirone, an antianxiety agent, is different from benzodiazepines and barbiturates in chemical structure, clinical pharmacology, and binding to γ -aminobutyric receptors. It has a strong affinity for dopamine and serotonin type 1 receptors. Buspirone has no muscle relaxant effect and no sedative property and does not seem to affect reaction time, vigilance, psychomotor speed, or memory function in elderly subjects (1), so it appears interesting for use with elderly patients. Buspirone has already been used for agitation in demented patients (2). We report the effect of buspirone in three patients with "probable" dementia of Alzheimer's type (on the basis of the National Institute of Neurological and Communicative Disorders and Stroke and the Alzheimer's Disease and Related Disorders Association criteria [3]) and generalized anxiety disorder (DSM-III-R) assessed by a semistructured interview with patient and caregiver. Patients had a moderate dementia of Alzheimer's type (mean Mini-Mental State score=14), no depressive disorders (DSM-III-R), and no history of psychiatric illness.

Ms. A, a 71-year-old woman, was anxious for 6-12 months. She experienced apprehensive expectation all the time, symptoms of motor tension, keyed-up feeling, and irritability. Fluoxetine, 20 mg/day p.o., was prescribed during 1 month without benefit and then was discontinued, and buspirone, 30 mg/day p.o., was introduced. Six weeks later her anxiety was lower. Her husband observed euphoria and pressured speech, as we did.

Ms. B, a 71-year-old woman, manifested depressed mood but no depressive disorder (as defined in DSM-III-R) and was treated by her general practitioner with fluoxetine, 20 mg/day p.o., over 3 weeks without benefit. When she came to us, Ms. B showed a generalized anxiety disorder of many months' duration with excessive worry, symptoms of motor tension, and keyed-up feeling. Buspirone, 30 mg/day p.o., was prescribed in addition to fluoxetine, which was continued at the same dosage. Two weeks later Ms. B manifested euphoria, pressured speech, disinhibition, and motor hyperactivity. Buspirone was discontinued by her husband and elation disappeared.

Mr. C, a 75-year-old man, had mood lability with crying, irritability, and manifestations of generalized anxiety disorder for about 6 months. Therapy with buspirone, 30 mg/day p.o., and fluoxetine, 20 mg/day p.o., was prescribed. Six weeks later, mood and manifestations of generalized anxiety disorder improved greatly and Mr. C displayed an elated mood with pressured speech.

Buspirone was not discontinued by Mr. C and Ms. A because their dependency was improved with euphoria.

Euphoria with pressured speech was observed in our three

patients treated with buspirone, 30 mg/day p.o., after fluoxetine treatment. They did not manifest delirium, sleep disorder, or manic disorder (DSM-III-R). Disinhibitory effects were observed in rats with buspirone and could be explained by a possible role of buspirone on dopamine receptors (4). Schweizer et al. (5) reported an antidepressant effect with high doses of buspirone. No depressive disorders were observed in our patients, and buspirone was prescribed at low dosage after fluoxetine. These patients suffered from dementia of Alzheimer's type in which serotonergic dysfunction was reported (6). Before proposing buspirone for the management of aggression in demented patients (2), the clinical feature of aggression might be specified.

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Fluoxetine and Prolonged Erection

SIR: Fluoxetine is reported to cause impotence and delayed ejaculation in several studies (1-3). Trazodone and phenelzine are the only antidepressants reported to cause priapism (4, 5). We report a case where fluoxetine caused prolonged erection and total loss of ejaculation. It is not a case of priapism since it occurred while the patient was being sexually stimulated and it resolved spontaneously.

Mr. A, a 41-year-old man, was seen after referral by his primary physician for labile affect and angry outbursts at family members. He met DSM-III-R criteria for major depressive disorder and was not psychotic. His only medication was alprazolam, 0.5 mg t.i.d. Fluoxetine was begun at 20 mg/day with partial success. It was increased to 40 mg/day, and his depression improved. After 2 weeks, he was engaging in sexual activity with his wife and developed a painful erection which lasted 2 hours without ejaculation and then resolved. The fluoxetine was discontinued and then restarted at 20 mg/day, but his depression recurred. A trial of fluoxetine with low-dose desipramine caused excessive anticholinergic effects. The desipramine was discontinued, and on the regimen of 20 mg/day of fluoxetine he had an episode of prolonged erection after intercourse with or-

gasm. The fluoxetine was stopped, and he was stabilized on bupropion and lithium.

This does not represent a case of priapism due to fluoxetine, but it is a case of prolonged erection which caused a clinically similar condition. It also is not a case of painful ejaculation, as was recently reported (6). Rather, it is a case of prolonged painful erection after sexual stimulation. Penile erection and ejaculation are due to peripheral parasympathetic and α -adrenergic activity, and fluoxetine is an antidepressant with the lowest affinity for cholinergic and α -adrenergic receptors. The mechanism by which this occurred in our patient is perhaps due to CNS serotonin reuptake blockade. Serotonin has been hypothesized (through 5-HT_{1B} receptors) to be the CNS mechanism for the production of penile erections, and 5-HT_{1B} agonists and reuptake blockers (including trazodone, fenfluramine, fluoxetine, and zimelidine) have induced erections in rats and primates (7, 8). Physicians should be aware of this in prescribing fluoxetine to men.

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Treatment of Panic Disorder in Coronary Artery Disease

SIR: Cardiologists often see patients with coronary artery disease who continue to complain of chest pain despite seemingly adequate anti-anginal therapy. Two studies (1, 2) have suggested that 40%-50% of patients presenting in a cardiology clinic with atypical chest pain and coronary artery disease fit criteria for panic disorder. At least one study found that alprazolam could be of benefit in reducing anxiety and angina in patients with ischemic heart disease who were stabilized on propranolol (3). We report a case of coronary artery disease associated with continuing chest pain that was not responsive to anti-anginal therapy until discovery of a concomitant panic disorder that was treated with clonazepam.

Ms. A, a 61-year-old woman with a 4-year history of stable coronary artery disease, presented in a cardiology clinic complaining of as many as six chest pain episodes a

week for the past 6 months. Ms. A was referred to a psychiatry research study of panic disorder in patients with coronary artery disease as it was thought that there may have been a component of anxiety to Ms. A's chest pain. The screening interview revealed that Ms. A met criteria for panic disorder with a 6-month duration. In addition to chest pain, Ms. A was experiencing episodes of dizziness, numbness and tingling, fear of dying, nausea, and tachycardia.

As part of the study protocol, Ms. A was entered into a double-blind crossover trial of placebo and clonazepam. While taking placebo during the first 4 weeks of the trial, Ms. A experienced only a mild decrease in symptoms. Her mean score on the Hamilton depression scale was 20, which was down from a score of 32 at baseline. She continued to experience an average of four chest pain episodes a week, at least three of which were full-symptom panic attacks.

Ms. A was withdrawn from placebo over 2 weeks and placed on clonazepam. For the next 4 weeks Ms. A's average Hamilton score was 5. During the entire 4-week treatment period, she experienced no chest pains and no symptoms of panic disorder. At the end of her study participation, Ms. A was taking 2.5 mg of clonazepam per day and she had been maintained on this dosage for an additional 6 weeks. She was being followed by her cardiologist.

As far as we know, this is the first report of a response to antipanic treatment by a patient with known coronary artery disease and panic disorder. Psychiatrists and other physicians should bear in mind the possibility that panic disorder may coexist with established organic disease.

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Pregnancy and Clozapine

SIR: We present the following case as it highlights an interesting clinical situation associated with the use of clozapine over typical antipsychotics and expands the data regarding the use of psychopharmaceuticals in pregnancy.

Ms. A, a 30-year-old woman with a long history of treatment-resistant chronic undifferentiated schizophrenia, was prescribed clozapine for the past 11 years with notable improvement in her symptoms. She was in a stable monogamous relationship for the past 8 years.

Several years after initiation of treatment with clozapine, Ms. A ceased to remove her coat during office visits. When

questioned, she removed her outer garment to reveal a distended abdomen, stating that she believed herself to be about 6 months pregnant. After a careful discussion of the risks of clozapine exposure during gestation and of her preparedness for parenthood, the patient was adamant about carrying her child and remaining on clozapine. It is of note that on two prior occasions while taking clozapine this patient became pregnant against clinical recommendations, choosing to terminate these pregnancies.

Gestation was complicated by the development of gestational diabetes in the second trimester but otherwise proceeded without incident. Labor was induced by prostin gel at 38 weeks, due in part to Ms. A's inability to comply satisfactorily with diabetic dietary restrictions. Labor and delivery were without complications except for shoulder dystocia. The child, a boy, was born at 8 lbs 2 oz with Apgar scores of 7 at 1 minute and 9 at 5 minutes. It is of note that the patient experienced no exacerbation of her psychiatric illness throughout gestation, labor, or delivery.

Clozapine clearly offers benefits to many treatment-resistant schizophrenic patients (1), but along with these are some associated caveats. Clozapine does not increase prolactin (often associated with infertility) in the same way as do more typical antipsychotics (2). Sexually active patients prescribed clozapine may be more likely to conceive. Additionally, as clozapine treatment may allow for increased social functioning (1), patients may find themselves in stable relationships and desirous of children. This case highlights the need to monitor patients for the onset of pregnancy. Our patient's reluctance to remove her winter clothing might be seen as an exacerbation of psychotic thought processes or an attempt to cover the physical distortion of clozapine-associated weight gain; neither was, in fact, the case. As of December 1990, at least 14 women were known to have been exposed to clozapine during gestation with no known adverse sequelae in their newborns (3). This case also emphasizes the need for further research on the use of psychotropic medications in pregnancy.

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Carbamazepine and Plasma Levels of Clozapine

SIR: It is our aim to remind clinicians of the potential pharmacokinetic interaction between clozapine and carbamazepine.

During recent years clozapine has gained a widening clinical use as an atypical neuroleptic, especially among some of the patients resistant to traditional neuroleptics (1). In addition to agranulocytosis, various forms of epileptic convulsions do

occur more often during the use of clozapine than with the classical neuroleptics. As antiepileptic drugs can be used with clozapine, the epileptic convulsions do not necessarily compel physicians to stop the clinically beneficial use of clozapine.

Carbamazepine is one of the most widely used antiepileptic drugs. Recently it has also gained popularity in the treatment of lithium-resistant and rapid-cycling bipolar affective disorders, as well as in the treatment of various psychotic and behavioral disorders. Carbamazepine is known to be a potent inductor of liver microsomal enzyme systems regulating the inactivation of a variety of drugs. Carbamazepine can thus substantially decrease the plasma levels of neuroleptics as well as other drugs (2-4). Similarly, the abrupt stopping of carbamazepine can markedly increase the plasma levels of concomitantly used neuroleptics, which results in the appearance of sometimes serious side effects.

We wish to report our experience with two patients whose plasma levels of clozapine increased significantly after stopping the concomitant use of carbamazepine. The first patient was a 25-year-old schizophrenic man who had been administered clozapine, 800 mg/day, and carbamazepine, 600 mg/day, for several months. The second patient was a 36-year-old schizophrenic man with epilepsy, taking clozapine, 600 mg/day, and carbamazepine, 800 mg/day. After carbamazepine was discontinued, the plasma levels of clozapine of the first patient increased from 1.4 to 2.4 $\mu\text{mol/liter}$ and in the second patient from 1.5 to 3.0 $\mu\text{mol/liter}$. The increase in the plasma levels of clozapine occurred within 2 weeks and were well above the suggested therapeutic level of 1.1 $\mu\text{mol/liter}$ (0.35 mg/liter).

With the widening psychiatric use of carbamazepine, clinicians should be well aware of the potential dangers and inconveniences that the induction of liver microsomal enzyme by carbamazepine use can cause to the patients using neuroleptics and other drugs with serious side effects. It is our opinion that for this reason valproate or oxycarbamazepine should be chosen as the antiepileptic drug for the prevention of convulsions during treatment with clozapine or other neuroleptics.

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Anorexia Nervosa and Lower Vulnerability to Infections

SIR: Several clinical observations have shown a paradoxically lower prevalence of infections in patients with anorexia nervosa during severe denutrition episodes (1).

In this prospective study, we investigated the possible cor-

relation between weight variations, total immunoglobulin amounts, and natural autoantibodies (2) that have been shown to interact with both self antigens and non-self-external pathogens in the sera of 10 female inpatients with anorexia nervosa according to DSM-III-R criteria. The evaluation was made upon admission and, after clinical improvement (less than two standard deviations of the ideal standard weight for age and height [3]), at hospital discharge. We determined the total IgM and IgG amounts by nephelometry as well as IgM and IgG autoantibody levels against several autoantigens including IgG F(ab')₂ by enzyme-linked immunosorbent assay (4). We compared the total IgG and autoantibody levels in anorexia nervosa patients to a control group of 10 healthy age-matched females.

We observed that total IgM amounts were significantly increased at admission and became normal at discharge. Inversely, total IgG amounts were significantly low upon admission and increased at discharge. In parallel to the total IgM increase, the IgM natural autoantibody activity was higher in patients, though not significantly. Interestingly, the only autoantibody activity we found significantly modified was the IgM anti-IgG F(ab')₂, which was decreased in correlation with the weight loss: the more deficient the weight was upon admission, the lower this activity was (Spearman rank correlation coefficient: $r=-0.69$).

In order to explain the lower prevalence of infections in patients with anorexia nervosa the results we obtained could suggest two possible levels of natural humoral immunity involvement:

1. A first line of nonspecific defense mechanism related to the increase of both total and natural autoantibody IgM that may enhance monocytes phagocytosis of pathogenic agents (2).

2. An IgG-dependent specific defense mechanism related to the IgM anti-IgG F(ab')₂ activity. In a recent study, it was demonstrated in normal serum that IgM regulates IgG activity (5). In anorexia nervosa, the decreased IgM anti-IgG F(ab')₂ may reflect the freeing of IgG antibody activities normally inhibited by IgM binding.

It is likely that the low antigenic stimulation by digestive tracts could interfere with these two mechanisms.

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Violent Antisocial Behavior and Wisconsin Card Sorting Test Performance in Cocaine Addicts

SIR: To further explore the association between frontal lobe dysfunction and antisocial and violent behavior, we administered the Computerized Diagnostic Interview Schedule (C-DIS) (1), which includes an assessment of antisocial personality disorder as defined in DSM-III-R, and the Wisconsin Card Sorting Test, a measure of frontal lobe performance (2), to 45 male crack cocaine-addicted inpatients on a locked substance abuse research unit. Subjects underwent the Structured Clinical Interview for DSM-III-R (SCID); none met DSM-III-R criteria for axis I diagnoses other than substance use disorder. The average age of the patients sampled was 34.0 (SD=7.4). The C-DIS revealed that 14 (31%) of the sample met DSM-III-R criteria for antisocial personality disorder.

On the basis of the number of violent items endorsed on the C-DIS before and after age 15, we assigned the 14 antisocial subjects to two groups: a high violence group (who endorsed at least six violent items) and a lower violence group (who endorsed no more than four violent items). A Mann-Whitney test revealed a significant difference between the two groups in the number of violent items endorsed (high violence mean=7.4, SD=1.6; low violence mean=3.2, SD=1.2; $z=3.2$, $df=12$, $p=0.0012$). However, the two groups did not differ significantly on the total number of C-DIS antisocial items endorsed (high violence mean=29.8, SD=8.1; low violence mean=27.8, SD=3.5; $z=0.2$, $df=12$, $p=0.84$). There were no significant differences in patterns of use between the two groups. When we compared the Wisconsin Card Sorting Test performance of the two groups there was a significant difference between the two groups on the number of perseverative errors. The low violence antisocial group made significantly more perseverative errors (mean=24.0, SD=15.7) than the antisocial sample (mean=11.4, SD=4.4; $z=2.1$, $df=12$, $p=0.03$).

Though literature exists suggesting a relationship between frontal lobe dysfunction and antisocial behavior, a careful review leaves the association between specifically violent criminal behavior and frontal lobe dysfunction in doubt (3). The results of our study suggest that, at least in the cocaine-addicted population we studied, high violence individuals diagnosed with antisocial personality disorder seem to have significantly better Wisconsin Card Sorting Test performance than low violence antisocial patients. Investigators describe a relationship between frontal lobe function and one's ability to rapidly adjust responses to external cues from the environment (2). One might speculate, therefore, that individuals with violent antisocial personality disorder who perform well on the Wisconsin Card Sorting Test are perhaps more effective at reading cues in the environment, which allows them to maximize their potential gain from utilizing violent behavior. The recent increase of physical violence in certain urban areas across the United States related to crack cocaine use suggests the need for future investigations exploring the relationship between violent behavior and the neurocognitive profiles of this population.

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Suicidal Ideation and Young Adults

SIR: The article by Patrick J. Meehan, M.D., and associates (1) brings up an important issue, namely, the high prevalence of suicidal thoughts in a normal population. Of 694 college freshmen/women in the sample, 54% had ever considered suicide, 10% had attempted it, and 3% sought help as a result of such attempts. Data presented but not discussed (table 1 of Dr. Meehan and associates' article) show that 62% of women, compared with 44% of men, had ever considered suicide. Almost the same proportion of men (43%) and women (49%) had thought of specific ways to take their own lives. Another recent study corroborates these findings among high school students: 33% had thought of suicide, and 4.5% reported an attempt in the previous year (2).

When half or more of a sizable, typical cohort exhibits a phenomenon associated with psychiatric morbidity and mortality, how are we to view it? Not, I submit, by striving to eradicate suicidal thoughts per se, although a reduction in their prevalence might be a good thing.

Instead, we might view the phenomenon as fairly typical of a stressful transition. Although a sign of pain, suicidal ideation may indicate health as much as illness. The refusal of suicide suggests a commitment, an engagement with life which is stronger than passive acquiescence. In the psychiatric history, I have come to regard episodes of suicidal ideation with concern, but not qualms, in most cases. It may be no more, and no less, than a pause at the mirror of philosophy: Is this life worth living? Even Albert Camus, who called it "the one truly serious philosophical problem," remarked that suicide is rarely committed through reflection (3).

The literature of autobiography abounds with instances of suicidal ideation followed by an affirmation of life. Mark Twain wrote about putting a gun to his head at the age of 30: "Many times I have been sorry I did not succeed, but I was never ashamed of having tried" (4). A generation later in Freud's Vienna, Otto Rank, at age 20, went through a similar crisis and wrote: "Afterwards there grew in me the greatest lust for life and courage towards death" (5). William James confronted suicide at 28, and wrote at 54, "I take it that no man is educated who has never dallied with the thought of suicide" (6).

Many similar examples could be cited, i.e., Arthur Rubinstein, Graham Greene, Karen Horney . . . Is the episode a sign or harbinger of pathology? I submit that we do not know and should be slow to assume so. Many young people today—close to half—have suicidal thoughts at least once. Epidemiological study may shed light on the psychiatric and social significance of the phenomenon. Meanwhile, we should be cautious about putting suicidal ideation on a single continuum of disease and death. The phenomenon may have as much or more to do with affirmation of life and immunity to actual suicide. It may be a part of normal, healthy adult development.

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Dissociation and Eating

SIR: The article by Edward A. Walker, M.D., and colleagues (1) highlights the frequency with which dissociative symptoms occur in certain groups of medical and psychiatric patients. Besides chronic pelvic pain, significant dissociation is found in patients with pseudoseizures (2), prolonged physical and sexual abuse in childhood (3), and eating disorders (4). Significant dissociation is also found in nonpatient groups composed of non-traditional healers (5), prostitutes, and exotic dancers (6).

We would like to report another group in which significant dissociation exists. This group includes psychiatric patients who diet but inexplicably do not lose weight despite valiant efforts on their part. We have seen three such patients, in all of whom severe dissociative disorders were first suspected after their eating patterns were thoroughly investigated. Mr. A, who was just beginning therapy, had no weight loss despite vigorous exercise and eating only one meal daily. Subsequent investigation revealed multiple personality disorder, in which alter personality states were surreptitiously eating. Ms. B had been treated 7 years for borderline personality disorder. She failed to lose weight despite reports of eating only one small meal daily. It was discovered that she had multiple personality disorder and an alter personality who ate surreptitiously. Ms. C had been previously diagnosed with anorexia nervosa and borderline personality disorder. She failed to lose weight despite vigorous exercise and a severely restricted diet. Subsequent investigation revealed that she had an ego state which she labeled "the sleepwalker" who arose early in the morning and binged on high-caloric sweets.

If clinicians encounter such patients who fail to lose weight on severely restricted diets and this is confirmed by a thorough dietary history, we would like to suggest screening for dissociative disorders through use of the Dissociative Experiences Scale (7) and then administering the Structured Clinical Interview for Dissociative Disorders (8) for those with significant scores (over 20).

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Folie à Deux Involving a Dog

SIR: Since Pavlov, scientists and the public have become familiar with the phenomena of conditioned behavior. Dr. Robert Howard (1) reported a proposed folie à deux involving a dog. By his own admission the dog's behavior cannot be considered delusional but rather a behavioral response conditioned by its owner. I think it unfortunate that the *Journal* gives credibility to this report by publishing it. To any lay or professional reader who understands the relationship between a lonely old woman and her devoted dog, this story would appear quite straightforward and not a psychiatric diagnosis.

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Dr. Howard Replies

SIR: Anyone who has given the subject of induced psychosis any thought will have been puzzled as to the possible mechanism by which a nonpsychotic individual could come to share the delusional beliefs and experience the hallucinatory phenomena of a mentally ill cohabitee. Cases of folie simultanée, communiquée, or induite are easy to explain, since both sufferers were psychotic to start with. Nowhere, however, can I find a convincing theory to explain the appearance of psychotic behavior and thinking in a previously well person who lives in close proximity to a psychotic person. It has been suggested that the recipient learns abnormal behavior from a more dominant driving inducer through the mechanism of classical learning theory (1). The case of Ms. A and her dog seems to offer some support for this view, since from it we learn that a psychotic woman was able to condition her dog to act in a way that was consistent with the content of her delusions. If Dr. Metz knows of another "lonely old woman and her devoted dog" who are engaged in the bizarre set of behaviors described in the case report, or if he feels able to produce an explanation for the induction of psychosis in cases of folie à deux, then he certainly has something to write about.

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Isolated but Serious Acts of Self-Mutilation

SIR: In his letter on the topic of a proposed model for self-injurious behavior (1), Ronald Pies, M.D., suggested two broad types of self-injurious behavior. He characterized the first type as consisting of isolated but serious acts, such as autocastration, and stated that these are generally committed in early adulthood by schizophrenic or other psychotic patients. In our study of seven previously unreported cases of genital self-mutilation (2) we found only one case involving psychosis, and in a later case study on yet another nonpsychotic genital self-mutilator (3) we conjectured that the association between serious acts of self-mutilation and psychosis may have been exaggerated by selective reporting.

Equally important, it was clear that in a generally young penitentiary population, genital self-mutilators were older than expected, being mostly in their third or fourth decade (2, 3). The same is true of ocular autoenucleators (4) and in a small series of self-amputees (5), none of whom was psychotic. The suggested associations of serious isolated acts of self-mutilation with psychosis and with relative youth are therefore not confirmed by our experience or that of others. In their reply to Dr. Pies' letter, Winchel and Stanley (6) argue that it is too early for models of the type that Dr. Pies proposes. I would agree with them that the available data do not yet support a general unifying theory of self-injurious behavior.

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Dr. Pies Replies

SIR: I thank Dr. Conacher for calling my attention to his seven cases of genital self-mutilation, of which only one involved psychosis. However, I maintain that the literature as a whole supports the association between isolated, serious ("ab-lative") acts of self-injury and some form of psychosis. In their own review of 53 cases of self-mutilation, Greilsheimer and Groves (1) found that 87% of patients were believed to be psychotic at the time of the act; 27 of the 53 carried a diagnosis of schizophrenia. In a more recent review, Martin and Gattaz (2) also noted that the most frequent diagnoses in genital self-mutilation are schizophrenia and affective psychoses. In a report of four cases of self-enucleation, Jones (3) found a specific association with paranoid delusions, either as a result of a drug-related toxic psychosis or schizophrenia. This confirmed the earlier impression of MacLean and Robertson (4) that psychosis—usually in the context of schizophrenia—in addition to a severe disturbance in body image are necessary variables in the act of self-enucleation.

It seems probable that many cases of isolated, serious self-

injury go unreported or uninvestigated, and that our diagnostic conclusions are based on a small subpopulation. Dr. Conacher's "penitentiary population" is a still more select group and may not represent the diagnostic types seen in the general population or in routine psychiatric practice.

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Antisemitism and the Collective Unconscious

SIR: In their letter Drs. Gerhard Bengesser and Stephen Sokoloff (1) proposed that the Jungian concept of collective unconscious, as expressed in "negative archetypes," could explain the "intergenerational aspect of phenomena such as antisemitism." Such a universal explanation would be inconsistent with the fact that a persistent and murderous antisemitism, crossing not only generations but centuries, prevails only in Christian societies. Islamic antisemitism does not distinguish between Jews and other infidels (including Christians), nor is it murderous. At present it is merely political and expedient and exploits Christian religious antisemitism. Ancient pagan antisemitism was no more than the normal, frequent, intercommunal rivalry that prevailed all over the pagan world without the specificity or persistence of Christian antisemitism. We will not find the answers to questions like this unless we formulate the questions correctly.

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Drs. Bengesser and Sokoloff Reply

SIR: Different facts in different areas require a careful assessment and every religion or movement deserves that careful assessment. Jung did not deal with such problems, especially interdenominational differences. His classical archetypes are often compared to Plato's forms or ideas. Antisemitism could only be a pseudoarchetype. Our intention was merely to propose a mechanism for the transgenerational transmission of tendencies such as antisemitism. We speculated that not only a priori concepts but also historically developed myths could form the contents of the so-called collective unconscious. This might possibly make the persistence of irrational notions such as antisemitism comprehensible. Our aim was lastly to make a contribution to the eradication of antisemitism.

A justification for the speculative thinking in our letter is that the world is not totally explainable or discernible. Physical thinking today in various fields is dominated by this fact

(as the prevailing ideas of the so-called Copenhagen Interpretation of quantum theory and John Bell's theorem are showing), which is in contrast to the mechanistic and mostly atheistic scientific thought of the nineteenth century. As the world remains not altogether explainable or discernible, so all the more should some fields of psychology and psychiatry be open to quasi-metaphysical ideas. In the sense of Karl Jaspers, comprehension has to replace explanation.

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Efficacy of Brief Psychotherapy

SIR: I read with interest the article by Paul Crits-Christoph, Ph.D. (1), and found agreement with his stated reasons for trying to find ways to assess the actual practice of psychotherapy, such as concern over third-party agencies. However, Dr. Crits-Christoph's meta-analysis is of data from studies employing manual-based therapies, not from therapy as it is actually and optimally practiced. There is some acknowledgment of this in the paper, but I believe the dangerous implications which relate to the stated purpose of the study are not fully recognized. The phrase "highly controlled conditions" used in his article is often taken to connote validity or meaningfulness which may lead agencies to effect policies based on erroneous conclusions about the actual practice of dynamically informed psychotherapies.

These errors would result from a faulty premise that the use of therapy manuals ensures some measure of quality of service delivered. Adherence to manuals may, in fact, limit the quality of service. Manuals tend to represent dynamically informed therapy in the most simplified, easily reproducible, and measurable elements. This reduces psychotherapy to terms of the lowest common denominator. Proficient (not merely "experienced") psychotherapists strive for much higher standards. These often require creative skills that admittedly are difficult to control and measure but which are essential.

Creative skills may be included in the flexibility referred to by Dr. Crits-Christoph and may involve, for instance, the janusian and homospacial processes as described by Rothenberg (2). These processes allude to the essence of effective psychotherapy. H. Strupp and associates (unpublished panel session, 1991) have stressed the difference between adherence to a manual versus "effective adherence," stating that in the latter "something else is apparently going on." I believe the "something else" is a necessary ingredient quite separate from what is imparted by therapy manuals. Thus, a study measuring the effect of manual-based therapy says nothing about the effect of good psychotherapy.

Studies can be well designed without simplifying and homogenizing psychotherapy. Blatt (3) has shown how precise diagnoses derived from in-depth theoretically relevant study of patient data are instrumental in determining the relative effects of different treatment modalities. His review of the data from the Menninger Psychotherapy Research Project has pro-

duced important findings that contrast sharply with numerous previous reports that failed to discriminate among treatment types in terms of therapeutic outcome.

Furthermore, there have been conceptual and methodological developments in single-subject research that may offer valid and generalizable information about dynamically informed psychotherapy. Some investigators (4) have devised sound empirical strategies by conducting extraclinical analyses of clinical material from transcripts.

Clearly there is room for a number of useful approaches in psychotherapy research. The field, it seems, would do well to make better use of advances in inductive approaches to study psychotherapy as it is actually practiced.

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Dr. Crits-Christoph Replies

SIR: Dr. Testani's comments usefully highlight a point that I had not made clear enough in my article: that treatment manuals by themselves do not guarantee a high quality of service. However, the statements in his letter that studies employing treatment manuals reduce "psychotherapy to terms of the lowest common denominator" and that "a study measuring the effect of manual-based therapy says nothing about the effect of good psychotherapy" reflect a misunderstanding of the nature of state-of-the-art psychotherapy efficacy research employing treatment manuals.

First of all, treatment manuals need not and should not be "cookbooks" that are overly simplified versions of therapy that compromise the richness of actual clinical practice. In fact, I believe that the process of developing a treatment manual that includes careful observation of the treatment process and more precise communication about how to conduct treatment optimally and how to modify the treatment for circumstances that emerge with difficult patients will in the long run tend to help improve the quality of treatment actually delivered by practitioners.

In addition to considering the quality of a manual, it is im-

portant to recognize that the essence of psychotherapy efficacy research is in the standardization of treatment, not simply the use of a manual. Standardization involves many steps designed to ensure that the treatment is conducted in an optimal way (i.e., from the perspective of the experts who developed the treatment). These steps should include 1) selection of therapists based upon competency evaluations, 2) training of therapists in the treatment manual using both competence and adherence criteria for evaluation, and 3) ongoing clinical supervision or competence monitoring during a clinical trial. Clearly, a study that employs a treatment manual but does not implement the above procedures is an inadequate test of the potential effects of the therapy.

Even if manuals are used in conjunction with careful selection, training, supervision, and competence monitoring, might they still result in an overly simplified and rigid therapy? Our experience at the Center for Psychotherapy Research at the University of Pennsylvania, where we have completed three outcome studies using a manual guided brief dynamic therapy and have four additional trials in their early stages, is that they do not. The standardization steps in the research process serve to exclude therapists who adhere to other forms of dynamic therapy (e.g., confrontation approaches), have idiosyncratic styles not compatible with the manual (e.g., heavy reliance on self-disclosure), or do not effectively use the basic techniques of the manual (e.g., formulating and interpreting patients' core conflictual relationship themes). Within these general constraints, therapists have considerable flexibility to call upon their creative skills. Thus, we have "homogenized" therapy on some broad dimensions, ruling out techniques which are clearly out of bounds for the modality, but we have not removed the "something else" that Dr. Testani refers to as the essence of effective psychotherapy. On the contrary, our senior clinical supervisors see the process of selection and training in brief dynamic therapy as precisely the task of ensuring that the essence of effective psychotherapy is maintained and indeed emphasized. If a research program is finding that therapists are adhering to a manual but delivering poor quality treatment, this would indicate a problem with the specific manual per se, or with the training program, rather than a problem with the concept of manualized therapies.

I fully agree with Dr. Testani that other research strategies are important and can provide information about psychotherapy that manual-guided outcome studies can not provide. However, the evaluation of standardized treatments in clinical trials will continue to be a recommended approach to efficacy research. In regard to brief dynamic therapy, let's hope that such standardized treatments are implemented in a way that preserves the creativity and flexibility that effective dynamic therapists display.

PAUL CRITS-CHRISTOPH, PH.D.
Philadelphia, Pa.

Reprints of letters to the Editor are not available.

Corrections

In the letter "Panic Disorder and Suicidal Ideation" by Giovanni A. Fava, M.D., et al. (October 1992, p. 1412), the second sentence should end on the eighth line as follows: "SD=9.5 years). They all [instead of 'five of whom'] satisfied the DSM-III-R criteria . . .".

Tables 1 and 2 of "Regional Cerebral Glucose Metabolism in Bulimia Nervosa" by Paul J. Andreason, M.D., et al. (November 1992, pp. 1509 and 1510) present normalized data; the units of measure given in these tables ($\mu\text{mol/minute per } 100 \text{ g of tissue}$) do not apply.



American Psychiatric Association Psychiatric Placement Service

CONNECTICUT

Private group practice in southeastern CT is currently seeking a psychiatrist with a strong psychopharmacology background for an expanding outpatient psychiatric practice. Inpatient work is also available. Initial annual salary with a substantially higher compensation for partnership. Located one hour from New Haven, Hartford and Providence. #27

GEORGIA

Thriving group practice is looking for a general psychiatrist to join a multi-disciplinary team in providing inpatient and outpatient services. Physician will see psychiatric inpatients, consult on the alcohol and drug unit, and develop and outpatient practice to follow-up on hospital discharges. Dominant group in the area offering a wide referral base and the opportunity to make a substantial income - salary plus percentage. Medium sized city just 90 miles from Atlanta. #51

MASSACHUSETTS

Two academic positions available: Sr. Psychiatrist to head 250 bed state hospital - 80% administrative, 20% clinical/teaching/research; Unit Directorship at a university affiliated general/medical hospital - duties include program development and implementation, supervision/teaching, and direct clinical work. As university employees, both individuals would receive a competitive salary and be eligible for the full university benefit package which includes malpractice insurance, medical/dental coverage, generous leave policies, tax sheltered annuity, state retirement program and tuition benefits for self and family. #3

MISSISSIPPI

Adult and Child Psychiatrist Big city psychiatric practice without the hassles of the big city at the largest non-metropolitan hospital in the United States. Comprehensive psychiatric programs to include 62 beds for adult and adolescents, partial hospitalization and various outpatient services. #36

NEW HAMPSHIRE

Medical Director provides psychiatric services and participates in the clinical work for CMHC which offers treatment to both the chronically mentally ill and the general population in freestanding clinics located throughout the area. Comprehensive system with variety of services provided. #14

NEW JERSEY

Community based teaching hospital with comprehensive inpatient and outpatient mental health services offers following: Medical Dir. Adult IP Services - 30 bed, acute, open unit for patients ages 18 to geriatrics. Heads multi-disciplinary team, ensuring therapeutic milieu, and supervises clinical staff (caseload of 6-8 patients). Medical Director of Ambulatory Mental Health Services - develops and implements clinical policy, supervises psychiatrist and others, maintains access to and quality of care for a continuum of programs (outpatient, partial care, emergency/screening, etc.) with annual volume of 40,000 visits. Excellent compensation package. #12

OHIO

Large CMHC is currently looking for staff psychiatrists to provide psychiatric leadership for their many multi-disciplinary teams. Work would be in the area of outpatient, inpatient, or crisis intervention services. Broad base service delivery system provides interested psychiatrists with the opportunity for

variety within their practice. Professional isolation is not a problem here given the large treatment team staff which includes other psychiatrists. The center is located in a beautiful suburb in one of Ohio's fastest growing cities, Cleveland. The community offers safety, affordable housing, good schools and easy access to city life. Excellent compensation package. Call PPS for more details. #37.

PENNSYLVANIA

Northwestern PA - Medical Director, Outpatient Community Psychiatric Services, sought by a 580 bed general hospital. Opportunity to assist in shaping new system of delivery of CMH services. Additional opportunity for limited private practice. Academic appointment possible for qualified candidate. Must be BC with leadership experience. Excellent salary/benefit package with relocation offered. Located in Erie, a city rich in recreational opportunities. Low cost of living, low crime rate, with excellent schools. Close proximity to Pittsburgh, Cleveland, Buffalo, and Toronto. #29

TEXAS

Two positions available at large CMHC in city suburb: Medical Directorship involving administrative, supervisory, and clinical work; and a Clinical Staff position involving supervision, medication management and C/L work with non-medical staff. Staff of 190 FTE's provides a variety of services including work with dually diagnosed (SA/MI) and mentally retarded. Ideal person will enjoy public sector work in a busy atmosphere at a center that emphasizes psychosocial rehabilitation and interagency collaboration. The area has two universities and three recreational lakes and is in the fastest growing county in Texas! #54

For more information on these and other opportunities available through the APA'S PPS, contact Rebecca Kilmer or Arlis Richardson at (202)682-6108 or write to the Psychiatric Placement Service, American Psychiatric Association, 1400 K Street, NW, Washington, DC 20005.

Calendar

(Continued from page A16)

Bridgewater. Contact: George Sigel, M.D., Bridgewater State Hospital, 20 Administration Rd., Bridgewater, MA 02324; 617-727-6086, ext 388.

April 1-3, West Coast Neuropsychology Conference, "Neuropsychology With Children: Assessment and Management," University of California, San Diego. Contact: Cass Jones, Professional Conference Management, 7916 Convoy Court, San Diego, CA 92111; 619-565-9921 (tel), 619-565-9954 (fax).

April 6-19, international symposium, "Folk Medicine of Ireland" (21 hours of CME category I and 3 hours of category II credit available), sponsored by the Southern California Neuropsychiatric Institute, Dublin. Contact: Ann McCormick, SCNPI, 6794 La Jolla Blvd., La Jolla, CA 92037; 619-454-2102 (tel), 619-454-2104 (fax).

April 18, Fifth Annual Symposium, "Treatment of Headaches and Facial Pain," New York Headache Center, New York. Contact: Alexander Mauskop, M.D., Director, New York Headache Center, 301 East 66th Street, New York, NY 10021; 212-794-3550.

April 22-25, 11th Annual Symposium in Forensic Psychiatry, American College of Forensic Psychiatry, Santa Fe, N.Mex. Contact: Ed Miller, Executive Director, 26701 Quail Creek, Suite 295, Laguna Hills, CA 92656; 714-831-0236.

April 23-25, Ninth Annual Conference, "Demonstrating and Sharing Integrative Therapies," Society for the Exploration of Psychotherapy Integration, New York. Contact: Saul D. Raw, C.S.W., 133 Lincoln Place, Brooklyn, NY 11217-3605; 718-638-9526.

April 24-25, conference, "The Cutting Edge 1993: Treatment of Severe Personality Disorders," University of California, San Diego. Contact: Cass Jones, Professional Conference Management, 7916 Convoy Court, San Diego, CA 92111; 619-565-9921 (tel), 619-565-9954 (fax).

April 30-May 1, conference, "Women," Cambridge Hospital, Harvard Medical School, Wellesley College, Boston. Contact: Judy Reiner Platt, Ed.D., Cambridge Hospital, 1493 Cambridge Street, Cambridge, MA 02139; 617-864-6165.

MAY

May 5-9, 11th Scientific Meeting, "Chaos and/or Community," A.K. Rice Institute, Los Angeles. Contact: Nancy Angelo, A.K. Rice Institute, P.O. Box 1776, Jupiter, FL 33468-1776; 407-744-1350 (tel), 407-744-5998 (fax).

May 19-23, meeting, American Back Society, Buffalo. Contact: Aubrey A. Swartz, M.D., Director, 2647 East 14th Street, Suite 401, Oakland, CA 94601; 510-536-9929 (tel), 510-536-1812 (fax).

May 22-27, 146th Annual Meeting, American Psychiatric Association, San Francisco. Contact: George Campbell, Director, Meetings Management, APA, 1400 K Street, NW, Washington, DC 20005; 202-682-6193.

May 24, satellite meeting (held in conjunction with the APA annual meeting), American Society of Clinical Psychopharmacology, San Francisco. Contact: Peter Ross, P.O. Box 2257, New York, NY 10116-2257; 212-268-4260 (tel), 212-268-4434 (fax).

May 30-June 2, 16th Annual Meeting of the Canadian College of Neuropsychopharmacology, CCNP and the British Association of Psychopharmacology, Montreal. Contact: Dr. S.N. Young, Dept. of Psychiatry, McGill University, 1033 Pine Ave., W, H3A 1A1 Montreal, Que., Canada; 514-398-7317 (tel), 514-398-4370 (fax).

JUNE

June 9-11, international conference, "Chronic Diseases and Changing Care Patterns in an Ageing Society," Netherlands Society for Public Health and Science, University of Amsterdam, Amsterdam. Contact: Dr. Trudi van den Bos or Wien Limburg, Institute of Social Medicine, University of Amsterdam, Meibergdreef 15, 1105 AZ Amsterdam, The Netherlands; 31-20-5664707 (tel), 31-20-6912401 (fax).

June 18-19, conference, "Child Psychotherapy," Cambridge Hospital, Harvard Medical School, Boston. Contact: Judy Reiner Platt, Ed.D., Cambridge Hospital, 1493 Cambridge Street, Cambridge, MA 02139; 617-864-6165.

Books Received

- Practicing Psychotherapy: A Casebook**, by Michael Franz Basch. New York, Basic Books, 1992, 204 pp., \$30.00.
- Dream Portrait: A Study of Nineteen Sequential Dreams as Indicators of Pretermination**, by Alma H. Bond, Daisy Franco, and Arlene Kramer Richards. Madison, Conn., International Universities Press, 1992, 177 pp., no price listed.
- The Evolution of Character: Birth to Eighteen Years: A Longitudinal Study**, by Sylvia Brody and Miriam G. Siegel. Madison, Conn., International Universities Press, 1992, 553 pp., \$70.00.
- Existential/Dialectical Marital Therapy: Breaking the Secret Code of Marriage**, by Israel W. Charney. New York, Brunner/Mazel, 1992, \$34.95.
- Molecular Basis of Neurology**, edited by P. Michael Conneally, Ph.D. Boston, Blackwell Scientific, 1993, 273 pp., \$39.95 (paper).
- A Brilliant Madness: Living With Manic-Depressive Illness**, by Patty Duke and Gloria Hochman. New York, Bantam Books, 1992, 285 pp., \$22.50.
- Changing the Rules: A Client-Directed Approach to Therapy**, by Barry L. Dunoan, Andrew D. Soiovey, and Gregory S. Rusk. New York, Guilford Press, 1992, 284 pp., \$32.00.
- Treating PTSD: Cognitive-Behavioral Strategies**, edited by David W. Foy. New York, Guilford Press, 1992, 172 pp., \$45.00; \$16.95 (paper).
- Human Nature and Suffering**, by Paul Gilbert. New York, Guilford Press, 1992, 406 pp., \$19.95 (paper).
- Clinical Neurology**, 2nd ed., by David A. Greenberg, M.D., Ph.D., Michael J. Aminoff, M.D., F.R.C.P., and Roger P. Simon, M.D. Norwalk, Conn., Appleton & Lange, 1993, 321 pp., \$31.95 (paper).
- Conrad Ferdinand Meyer and Freud: The Beginnings of Applied Psychoanalysis**, by Alexander Grinstein. Madison, Conn., International Universities Press, 1992, 399 pp., \$49.50.
- Movement Disorders in Neurology and Neuropsychiatry**, edited by Anthony B. Joseph, M.D., and Robert R. Young, M.D. Boston, Blackwell Scientific, 1992, 748 pp., \$129.95.
- Engendered Lives: A New Psychology of Women's Experience**, by Ellyn Kaschak. New York, HarperCollins, 1992, 265 pp., \$25.00.
- Alzheimer's Disease: New Treatment Strategies**, edited by Zaven S. Khachaturian and John P. Blass. New York, Marcel Dekker, 1992, 228 pp., \$99.75.
- Guidelines for Neuroleptic Relapse Prevention in Schizophrenia**, edited by Werner Kissling. New York, Springer-Verlag, 1991, 166 pp., no price listed.
- Cognitive Neuropsychology in Clinical Practice**, edited by David Ira Margolin. New York, Oxford University Press, 1992, 548 pp., \$60.00.
- How Psychiatrists Look at Aging**, edited by George H. Pollock. Madison, Conn., International Universities Press, 1992, 244 pp., \$32.50.
- Metapsychology: Missing Links in Behavior, Mind, and Science**, by Sam S. Rakover. New York, Paragon House, 1992, 449 pp., \$35.00.
- Internalizing Disorders in Children and Adolescents**, edited by William M. Reynolds. New York, John Wiley & Sons, 1992, 336 pp., no price listed.
- Transgenerational Family Therapies**, by Laura Giat Roberto. New York, Guilford Press, 1992, 219 pp., \$26.95.
- Supportive Therapy for Borderline Patients: A Psychodynamic Approach**, by Lawrence H. Rockland. New York, Guilford Press, 1992, 308 pp., \$30.00.
- Counseling Chemically Dependent People With HIV Illness**, edited by Michael Shernoff. Binghamton, N.Y., Harrington Park Press, 1992, 172 pp., \$14.95.
- Psychoanalytic Perspectives on Women**, edited by Elaine V. Siegel. New York, Brunner/Mazel, 1992, 160 pp., \$21.95.
- Short-Term Anxiety-Provoking Psychotherapy: A Treatment Manual**, by Peter E. Sifneos. New York, Basic Books, 1992, 223 pp., \$30.00.
- Memory: Organization and Locus of Change**, edited by Larry R. Squire, Norman M. Weinberger, and Gary Lynch. New York, Oxford University Press, 1992, 423 pp., \$65.00.
- Psychology and Social Responsibility: Facing Global Challenges**, edited by Sylvia Staub and Paul Green. New York, New York University Press, 1992, 432 pp., \$57.50; \$19.50 (paper).
- Wisdom of the Elders: Honoring Sacred Native Visions of Nature**, by David Suzuki and Peter Knudtson. New York, Bantam Books, 1992, 272 pp., \$22.00.
- Freudian Fraud: The Malignant Effect of Freud's Theory on American Thought and Culture**, by E. Fuller Torrey. New York, HarperCollins, 1992, 362 pp., \$25.00.
- Ego Mechanisms of Defense: A Guide for Clinicians and Researchers**, by George E. Vaillant. Washington, D.C., American Psychiatric Press, 1992, 306 pp., no price listed.
- A Practical Guide to the Treatment of Bulimia Nervosa**, by Johan Vanderlinden, Jan Norré, and Walter Vandereycken. New York, Brunner/Mazel, 1992, 224 pp., \$22.95.
- Perturbing the Organism: The Biology of Stressful Experience**, by Herbert Weiner. Chicago, University of Chicago Press, 1992, 372 pp., \$35.00.

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THE AMERICAN JOURNAL OF PSYCHIATRY

Information for Authors

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The requirements stated below are in accordance with the International Committee of Medical Journal Editors. See Uniform Requirements for Manuscripts Submitted to Biomedical Journals, *N Engl J Med* 1991; 324:424–428.

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The *Journal* requires approval of manuscript submission by all authors in addition to transfer of copyright to the American Psychiatric Association so that the author(s) and the Association are protected from misuse of copyrighted material. A submission approval and copyright transfer form will be forwarded to authors along with a letter acknowledging receipt of their manuscript. Manuscripts will not receive a final decision until a completed form has been received in the editorial office.

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Disclosure of Commercial Interests

All forms of support, including drug company support, must be acknowledged in the author's footnote (see "Acknowledgments" under the Title Page section). Also, authors must disclose in their cover letter any commercial or financial involvements that might present an appearance of a conflict of interest in connection with the submitted article, including but not limited to institutional or corporate affiliations not already specified in the author's footnote, paid consultancies, stock ownership or other equity interests, and patent ownership. At the Editor's discretion, this information may be

shared with reviewers. Such involvements will not be grounds for automatic rejection of the manuscript. Should the article be accepted for publication, the Editor and the authors will consult on whether, and to what extent, this information should be included in the published article.

Patient Anonymity

Ethical and legal considerations require careful attention to the protection of a patient's anonymity in case reports and elsewhere. Identifying information such as names, initials, hospital numbers, and dates must be avoided. Also, authors should disguise identifying information when discussing patients' characteristics and personal history.

Informed Consent

Manuscripts that report the results of experimental investigation with human subjects must include a statement that informed consent was obtained after the procedure(s) had been fully explained. In the case of children, authors are asked to include information about whether the child's assent was obtained.

Review Process

All papers are reviewed to determine the originality, validity, and importance of content and conclusions. In addition to the regular review process, peer review for statistical content may be required for some manuscripts. This will be determined by the *Journal's* Statistical Editors. Authors will be sent reviewer comments that are judged to be useful to them. All reviewers remain anonymous. Once the Editor has made a final decision on a paper, the authors will be informed.

SUBMISSION OF MANUSCRIPTS

The original manuscript and four copies should be submitted to John C. Nemiah, M.D., Editor, *American Journal of Psychiatry*, 1400 K St., N.W., Washington, DC 20005. Telephone: (202) 682-6020. FAX: (202) 682-6016. All correspondence will be sent to the first-named author unless otherwise specified. Papers should be accompanied by a cover letter indicating that the paper is intended for publication, stating the number of figures, and specifying for which section of the *Journal* it is being submitted (i.e., Special Article, Regular Article, or Clinical and Research Report); papers that do not meet the requirements (including word count) for one of the types of articles specified in the next section will be returned unreviewed.

Authors will be notified of the receipt of their paper and the number assigned to it. This number must be included in all further correspondence. It is imperative that the correspond-

INFORMATION FOR AUTHORS

ing author of submitted papers notify the *Journal* of changes of address. Manuscripts reviewed by the *Journal* will not be returned to authors except upon special request. Authors must make this request in their original submission letter and include a self-addressed, postage-paid envelope.

Case Reports

Case reports (single or series) should be submitted as Letters to the Editor. All case reports will be peer reviewed. Reports of successfully treated patients must include data on the number of patients treated unsuccessfully by the same method, with an indication of the temporal order of the successes and failures.

Annual Meeting Papers

Papers may be submitted before the annual meeting, but they cannot be published until after the meeting. They must be accompanied by a statement that they are in final form. These papers receive the same peer review as other papers and must meet the requirements for one of the types of articles specified in the next section. The *Journal* no longer maintains right of first refusal for annual meeting papers.

TYPES OF ARTICLES

Special Articles

These are usually overview articles that bring together important information on a topic of general interest to psychiatry. Authors who have ideas for such articles are advised to check with the Editorial Office to ensure that a similar work has not already been submitted. Special Articles may not exceed 7,500 words, including an abstract of no more than 250 words, references, tables, and figures (to determine word equivalence, see section on Tables and Figures). This section is not intended to be a forum for the presentation of new data.

Regular Articles

Regular Articles are original communications of scientific excellence in psychiatric medicine and advances in clinical research, containing new data derived from a sizable series of patients. Regular Articles may not exceed 3,800 words, including an abstract of no more than 250 words, references, tables, and figures (to determine word equivalence, see section on Tables and Figures).

Clinical and Research Reports

Clinical and Research Reports present 1) data from pilot or uncontrolled studies with suggestive findings warranting further, more definitive investigation, 2) worthwhile replication studies, and 3) clinical studies involving a small number of patients. Essays, program descriptions, literature reviews, and case reports do not meet the criteria for this section. These articles may not exceed 1,300 words, including an abstract of no more than 60 words, references, tables, and figures (to determine word equivalence, see section on Tables and Figures).

Other Sections

Letters to the Editor. Brief letters (maximum of 500 words, including references; no tables or figures) will be considered

if they include the notation "for publication." A letter must be signed by all of its authors; three copies are required. Case reports should be submitted as Letters to the Editor. Case reports will be peer reviewed. Letters critical of an article published in the *Journal* must be received within 6 weeks of the article's publication; letters from outside the United States must be received within 12 weeks. Letters received after the deadline will not be considered; those accepted for publication will be sent to the authors for reply. Such letters must include the title and author of the article and the month and year of publication. Letters that do not meet these specifications will be returned unreviewed. The *Journal* will notify authors about the disposition of their letters. All accepted letters will be edited; proofs will not be sent to authors for approval. Reprints are not available.

Book Forum. Books for review may be sent to the Book Forum Editor, Nancy C. Andreasen, M.D., Ph.D., Director, Mental Health Clinical Research Center, University of Iowa Hospitals and Clinics, 200 Hawkins Dr., Iowa City, IA 52242. Book reviews are usually solicited by the Book Forum Editor. Authors interested in reviewing a particular book should communicate directly with Dr. Andreasen. Reprints of reviews are not available.

TYPING AND ARRANGING THE PAPER

All parts of the manuscript or letter to the Editor, including case reports, quotations, references, and tables, must be double-spaced throughout. Manuscripts must be typed in upper- and lowercase on one side only of 8.5×11 inch nonerasable bond paper. All four margins must be 1.5 inches. The manuscript should be arranged in the following order, with each item beginning a new page: 1) title page, 2) abstract, 3) text, 4) references, and 5) tables and/or figures. All pages must be numbered. The original and four copies of a manuscript, including tables and figures, should be submitted.

STYLE SPECIFICATIONS

Title Page

Title. The title should be informative and as brief as possible. Two-part titles should be avoided.

Byline. Authors listed in the byline should be limited to principal researchers and/or writers; collaborators may be acknowledged in a footnote. Authors' first names are preferred to initials. Degrees should be included after each author's name. The *Journal* subscribes to the Uniform Requirements for Manuscripts Submitted to Biomedical Journals (N Engl J Med 1991; 324:424-428) for authorship summarized here:

Authorship

All persons designated as authors should qualify for authorship. Each author should have participated sufficiently in the work to take public responsibility for the content.

Authorship credit should be based only on substantial contributions to (a) conception and design, or analysis and interpretation of data, and (b) drafting the article or revising it critically for important intellectual content and on (c) final approval of the version to be published. Conditions (a), (b), and (c) must all be met. Participation solely in the acquisition

of funding or the collection of data does not justify authorship. General supervision of the research group is also not sufficient. Any part of an article critical to its main conclusions must be the responsibility of at least one author.

Only those with key responsibility for the material in the article should be listed as authors; others contributing to the work should be recognized separately. Editors may require authors to justify the assignment of authorship.

Previous presentation. If the paper has been presented at a meeting, please give the name of the meeting, the location, and the inclusive dates.

Location of work and address for reprints. Provide the department, institution, city, and state where the work was done. Include a full address for the author who is to receive reprint requests.

Acknowledgments. Grant support should be acknowledged in a separate paragraph and should include the full name of the granting agency and grant number. See instructions for Disclosure of Commercial Interests. The *Journal* does not allow acknowledgment of persons involved with the preparation or typing of manuscripts. Acknowledgment of individuals involved with the scientific content of the work should not exceed four typed lines. Drug company support of any kind must be acknowledged.

Abstract

Authors of review articles must include the following information, under the headings indicated: **Objective**—the primary purpose of the review article; **Method**—data sources, study selection (the number of studies selected for review and how they were selected), data extraction (rules for abstracting data and how they were applied); **Results**—methods of data synthesis, key findings; and **Conclusions**—including potential applications and research needs. Authors of research articles must include **Objective**—questions addressed by the study; **Method**—design of the study, setting (location and level of clinical care), patients or participants (manner of selection and number who entered and completed the study), interventions (if any), main outcome measures (primary study outcome measure as planned before data collection); **Results**—key findings; and **Conclusions**—including direct clinical applications. Other types of articles, including Clinical and Research Reports, should include unstructured abstracts.

The abstract is a single paragraph no longer than 250 words for Special Articles and Regular Articles and no longer than 60 words for Clinical and Research Reports.

Text

Research design and statistics. The following information regarding research design should be included: 1) a clearly stated hypothesis, 2) the names of the statistical tests used, 3) whether tests were one- or two-tailed, and 4) what test was used for each set of data. Standard deviations, rather than standard errors of the mean, are required. Statistical tests that are not well known should be referenced. All significant and important nonsignificant results must include the test value, degree(s) of freedom, and probability. For example, "The analysis of variance indicated that those who abstained from coffee had significantly higher course grades than those who did not abstain ($F=4.32$, $df=3$, 17 , $p<0.05$).\" Reviewers will evaluate the appropriateness of the analyses.

Abbreviations. Spell out all abbreviations (other than those

for units of measure) the first time they are used. Idiosyncratic abbreviations should not be used.

Drugs. Generic rather than trade names of drugs should be used. Trade or manufacturers' names are used only if the drug or equipment is experimental or unavailable in this country or if such information is crucial to the evaluation of the results or replication of the study.

References

References are numbered and listed by their order of appearance in text; the text citation is followed by the appropriate reference number in parentheses. Do not arrange the list alphabetically. References in tables and figures are numbered as though the tables and figures were part of the text.

References should be restricted to closely pertinent material. **Accuracy of citation is the author's responsibility.** References should conform exactly to the original spelling, accents, punctuation, etc. Authors should be sure that all references listed have been cited in text.

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Type references in the style shown below, double-spaced throughout. List all authors; do not use "et al." Abbreviations of journal names should conform to the style used in *Index Medicus*; journals not indexed there should not be abbreviated.

1. Noyes R Jr, DuPont RL Jr, Pecknold JC, Rifkin A, Rubin RT, Swinson RP, Ballenger JC, Burrows GD: Alprazolam in panic disorder and agoraphobia, results from a multicenter trial, II: patient acceptance, side effects, and safety. *Arch Gen Psychiatry* 1988; 45:423-428
2. Kaplan HI, Sadock BJ (eds): *Comprehensive Textbook of Psychiatry*, 4th ed, vol 2. Baltimore, Williams & Wilkins, 1985
3. Fyer AJ, Manuzza S, Endicott J: Differential diagnosis and assessments of anxiety: recent developments, in *Psychopharmacology: The Third Generation of Progress*. Edited by Meltzer HY. New York, Raven Press, 1987

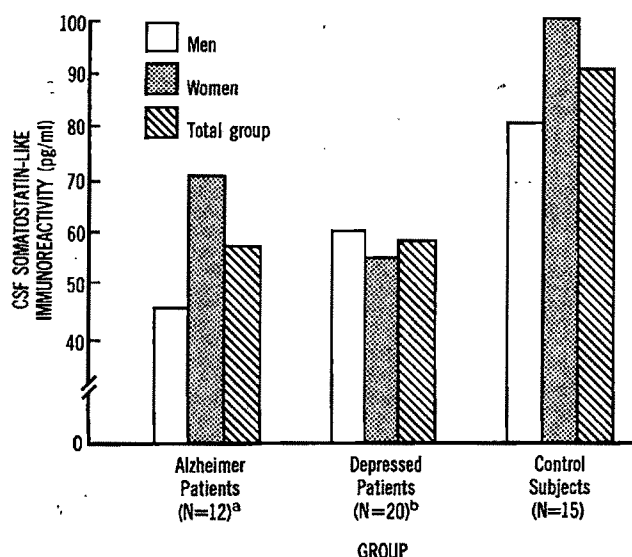
TABLES AND FIGURES

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Tables

Tables should be double-spaced, no wider than 120 typewriter characters, including spaces, and no longer than 70 lines. Values expressed in the same unit of measurement should read down, not across; when percentages are given, the appropriate numbers must also be given.

FIGURE 1. Mean CSF Somatostatin Levels of Patients With Alzheimer's Disease, Older Depressed Patients, and Age-Matched Control Subjects



^aSignificant difference between men and women ($t=1.81$, $df=10$, $p<0.05$) and between the total Alzheimer's disease group and the total control group ($t=2.49$, $df=25$, $p<0.01$).

^bSignificant difference between the total depressed group and the total control group ($t=2.75$, $df=33$, $p<0.005$).

Figures

Figures are considered as text and are subject to revision by the authors upon recommendation of the Editors. Figures should, however, be professionally prepared. Glossy or other camera-ready prints should accompany the submitted manuscript. Computer-generated figures that do not meet quality printing standards will be returned for revision. All figure titles and footnotes should be typed and sent together on a separate page.

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2. The heading for the vertical axis of a graph should run vertically along the axis, not horizontally at the top or bottom. Headings for the horizontal axis should appear below that axis, not at the top of the graph.

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BRIEF SUMMARY

ZOLOFT[®] (sertraline HCl)

INDICATIONS AND USAGE: ZOLOFT (sertraline hydrochloride) is indicated for the treatment of depression.

CONTRAINDICATIONS: None known. **WARNINGS:** In patients receiving another serotonergic reuptake inhibitor drug in combination with a monoamine oxidase inhibitor (MAOI), there have been reports of serious, sometimes fatal, reactions including hyperthermia, rigidity, myoclonus, autonomic instability with possible rapid fluctuations of vital signs, and mental status changes that include extreme agitation progressing to delirium and coma. These reactions have also been reported in patients who have recently discontinued that drug and have been started on an MAOI. Some cases presented with features resembling neuroleptic malignant syndrome. Therefore, it is recommended that ZOLOFT (sertraline hydrochloride) not be used in combination with an MAOI, or within 14 days of discontinuing treatment with an MAOI. Similarly, at least 14 days should be allowed after stopping ZOLOFT before starting an MAOI.

PRECAUTIONS General: Activation of Mania/Hypomania - During premarketing testing, hypomania or mania occurred in approximately 0.4% of ZOLOFT (sertraline hydrochloride) treated patients. Activation of mania/hypomania has also been reported in a small proportion of patients with Major Affective Disorder treated with other marketed antidepressants.

Weight Loss: Significant weight loss may be an undesirable result of treatment with sertraline for some patients, but on average, patients in controlled trials had minimal, 1 to 2 pound weight loss, versus smaller changes on placebo. Only rarely have sertraline patients been discontinued for weight loss. **Seizure:** ZOLOFT has not been evaluated in patients with a seizure disorder. These patients were excluded from clinical studies during the product's premarket testing. Accordingly, like other antidepressants, ZOLOFT should be introduced with care in epileptic patients. **Suicide:** The possibility of a suicide attempt is inherent in depression and may persist until significant remission occurs. Close supervision of high risk patients should accompany initial drug therapy. Prescriptions for ZOLOFT should be written for the smallest quantity of tablets consistent with good patient management, in order to reduce the risk of overdose. **Weak Urinary Effect:** ZOLOFT is associated with a mean decrease in serum uric acid of approximately 7%. The clinical significance of this weak uricosuric effect is unknown, and there have been no reports of acute renal failure with ZOLOFT. **Use in Patients with Concomitant Illness:** Clinical experience with ZOLOFT in patients with certain concomitant systemic illness is limited. Caution is advisable in using ZOLOFT in patients with diseases or conditions that could affect metabolism or hemodynamic responses. ZOLOFT has not been evaluated or used to any appreciable extent in patients with a recent history of myocardial infarction or unstable heart disease. Patients with these diagnoses were excluded from clinical studies during the product's premarket testing. However, the electrocardiograms of 774 patients who received ZOLOFT in double-blind trials were evaluated and the data indicate that ZOLOFT is not associated with the development of significant ECG abnormalities. ZOLOFT is extensively metabolized by the liver. The pharmacokinetics of ZOLOFT have not been studied in patients with significant hepatic dysfunction nor have patients with significant hepatic dysfunction been evaluated during treatment with ZOLOFT. Accordingly, ZOLOFT should be used with caution in such patients. Since ZOLOFT is extensively metabolized, excretion of unchanged drug in urine is a minor route of elimination. However, until the pharmacokinetics of ZOLOFT have been studied in patients with renal impairment and until adequate numbers of patients with severe renal impairment have been evaluated during chronic treatment with ZOLOFT, it should be used with caution in such patients. **Interference with Cognitive and Motor Performance:** In controlled studies, ZOLOFT did not cause sedation and did not interfere with psychomotor performance. **Information for Patients:** Physicians are advised to discuss the following issues with patients for whom they prescribe ZOLOFT. Patients should be told that although ZOLOFT has not been shown to impair the ability of normal subjects to perform tasks requiring complex motor and mental skills in laboratory experiments, drugs that act upon the central nervous system may affect some individuals adversely. Patients should be told that although ZOLOFT has not been shown in experiments with normal subjects to increase the mental and motor skill impairments caused by alcohol, the concomitant use of ZOLOFT and alcohol in depressed patients is not advised. Patients should be told that while no adverse interaction of ZOLOFT with over-the-counter (OTC) drug products is known to occur, the potential for interaction exists. Thus, the use of any OTC product should be initiated cautiously according to the directions of use given for the OTC product. Patients should be advised to notify their physician if they become pregnant or intend to become pregnant during therapy. Patients should be advised to notify their physician if they are breast-feeding an infant. **Laboratory Tests:** None. **Drug Interactions: Potential Effects of Coadministration of Drugs Highly Bound to Plasma Proteins:** Because sertraline is tightly bound to plasma protein, the administration of ZOLOFT (sertraline hydrochloride) to a patient taking another drug which is tightly bound to protein (e.g., warfarin, digitoxin) may cause a shift in plasma concentrations potentially resulting in an adverse effect. Conversely, adverse effects may result from displacement of protein-bound ZOLOFT by other tightly bound drugs. In a study comparing prothrombin time AUC (0-120 hr) following dosing with warfarin (0.75 mg/kg) before and after 21 days of dosing with either ZOLOFT (50-200 mg/day) or placebo, there was a mean increase in prothrombin time of 8% relative to baseline for ZOLOFT compared to a 1% decrease for placebo (p<0.02). The normalization of prothrombin time for the ZOLOFT group was delayed compared to the placebo group. The clinical significance of this change is unknown. Accordingly, prothrombin time should be carefully monitored when ZOLOFT therapy is initiated or stopped. **CNS Active Drugs:** In a study comparing the disposition of intravenously administered diazepam before and after 21 days of dosing with either ZOLOFT (50 to 200 mg/day escalating dose) or placebo, there was a 32% decrease relative to baseline in diazepam clearance for the ZOLOFT group compared to a 19% decrease relative to baseline for the placebo group (p<0.03). There was a 23% increase in t_{max} for desmethyldiazepam in the ZOLOFT group compared to a 20% decrease in the placebo group (p<0.03). The clinical significance of these changes is unknown. In a placebo-controlled trial in normal volunteers, the administration of two doses of ZOLOFT did not significantly alter steady-state lithium levels or the renal clearance of lithium. Nonetheless, at this time, it is recommended that plasma lithium levels be monitored following initiation of ZOLOFT therapy with appropriate adjustments to the lithium dose. The risk of using ZOLOFT in combination with other CNS active drugs has not been systematically evaluated. Consequently, caution is advised if the concomitant administration of ZOLOFT and such drugs is required. **Hypoglycemic Drugs:** In a placebo-controlled trial in normal volunteers, administration of ZOLOFT for 22 days (including 200 mg/day for the final 13 days) caused a statistically significant 16% decrease from baseline in the clearance of tolbutamide following an intravenous 1000 mg dose. ZOLOFT administration did not noticeably change either the plasma protein binding or the apparent volume of distribution of tolbutamide, suggesting that the decreased clearance was due to a change in the metabolism of the drug. The clinical significance of this decrease in tolbutamide clearance is unknown. **Atenolol - ZOLOFT (100 mg)** when administered to 10 healthy male subjects had no effect on the beta-adrenergic blocking activity of atenolol. **Microsomal Enzyme Induction:** Preliminary studies have shown ZOLOFT to induce hepatic microsomal enzymes. In clinical studies ZOLOFT was shown to induce hepatic enzymes minimally as determined by a small (5%) but statistically significant decrease in antipyrine half-life following administration of 200 mg/day for 21 days. This small change in antipyrine half-life reflects a clinically insignificant change in hepatic metabolism. **Electroconvulsive Therapy (ECT):** There are no clinical studies establishing the risks or benefits of the combined use of electroconvulsive therapy (ECT) and ZOLOFT. **Alcohol:** Although ZOLOFT did not potentiate the cognitive and psychomotor effects of alcohol in experiments with normal subjects, the concomitant use of ZOLOFT and alcohol in depressed patients is not recommended. **Carcinogenesis, Mutagenesis, Impairment of Fertility:** Lifetime carcinogenicity studies were carried out in CD-1 mice and Long-Evans rats at doses up to 40 mg/kg in mice (10 times on a mg/kg basis and the same on a mg/m² basis as the maximum recommended human dose) and at doses up to 40 mg/kg in rats (10 times on a mg/kg basis and 2 times on a mg/m² basis, the maximum recommended human dose). There was a dose-related increase in the incidence of liver adenomas in male mice receiving sertraline at 10-40 mg/kg. No increase was seen in female mice or in rats of either sex receiving the same treatments, nor was there an increase in hepatocellular carcinomas. Liver adenomas have a variable rate of spontaneous occurrence in the CD-1 mouse and are of unknown significance to humans. There was an increase in follicular adenomas of the thyroid in female rats receiving sertraline at 40 mg/kg; this was not accompanied by thyroid hyperplasia. While there was an increase in uterine adenocarcinomas in rats receiving sertraline at 10-40 mg/kg compared to placebo controls, this effect was not clearly drug related. Sertraline had no genotoxic effects, with or without metabolic activation, based on the following assays: bacterial mutation assay, mouse lymphoma mutation assay, and tests for cytogenetic aberrations *in vivo* in mouse bone marrow and *in vitro* in human lymphocytes. A decrease in fertility was seen in one of two rat studies at a dose of 80 mg/kg (20 times the maximum human dose on a mg/kg basis and 4 times on a mg/m² basis). **Pregnancy - Pregnancy Category B:** **Teratogenic Effects:** Reproduction studies have been performed in rats and rabbits at doses up to approximately 20 times and 10 times the maximum daily human mg/kg dose (4 to 4.5 times the mg/m² dose), respectively. There was no evidence of teratogenicity at any dose level. At doses approximately 2.5-10 times the maximum daily human mg/kg dose, sertraline was associated with delayed ossification in fetuses, probably secondary to effects on the dams. There are no adequate and well-controlled studies in pregnant women. Because animal reproduction studies are not always predictive of human response, this drug should be used during pregnancy only if clearly needed. **Non-teratogenic Effects:** There was also decreased neonatal survival following maternal administration of sertraline at doses as low as approximately 5 times the maximum human mg/kg dose. The decrease in pup survival was shown to be most probably due to *in utero* exposure to sertraline. The clinical significance of these effects is unknown. **Labor and Delivery:** The effect of ZOLOFT on labor and delivery in humans is unknown. **Nursing Mothers:** It

is not known whether, and if so in what amount, sertraline or its metabolites are excreted in human milk. Because many drugs are excreted in human milk, caution should be exercised when ZOLOFT is administered to a nursing woman. **Pediatric Use - Safety and effectiveness in children have not been established. Geriatric Use -** Several hundred elderly patients have participated in clinical studies with ZOLOFT. The pattern of adverse reactions in the elderly was similar to that in younger patients. **ADVERSE REACTIONS Commonly Observed:** The most commonly observed adverse events associated with the use of ZOLOFT (sertraline hydrochloride) and not seen at an equivalent incidence among placebo-treated patients were: gastrointestinal complaints, including nausea, diarrhea/loose stools and dyspepsia; tremor; dizziness; insomnia; somnolence; increased sweating; dry mouth; and male sexual dysfunction (primarily ejaculatory delay). **Associated with Discontinuation of Treatment:** Fifteen percent of 2710 subjects who received ZOLOFT in premarketing multiple dose clinical trials discontinued treatment due to an adverse event. The more common events (reported by at least 1% of subjects) associated with discontinuation included agitation, insomnia, male sexual dysfunction (primarily ejaculatory delay), somnolence, dizziness, headache, tremor, anorexia, diarrhea/loose stools, nausea, and fatigue. **Incidence in Controlled Clinical Trials:** The table that follows enumerates adverse events that occurred at a frequency of 1% or more among ZOLOFT patients who participated in controlled trials comparing titrated ZOLOFT with placebo. Most patients received doses of 50 to 200 mg per day.

Treatment-Emergent Adverse Experience Incidence in Placebo-Controlled Clinical Trials*					
(Percent of Patients Reporting)			(Percent of Patients Reporting)		
Adverse Experience	ZOLOFT (N=861)	Placebo (N=853)	Adverse Experience	ZOLOFT (N=861)	Placebo (N=853)
Autonomic Nervous System Disorders					
Mouth Dry	16.3	9.3	Hot Flashes	2.2	0.5
Sweating Increased	8.4	2.9	Fever	1.6	0.6
Cardiovascular			Back Pain	1.5	0.9
Palpitations	3.5	1.6	Metabolic and Nutritional Disorders		
Chest Pain	1.0	1.6	Thirst	1.4	0.9
Central and Peripheral Nervous System Disorders			Musculoskeletal System Disorders		
			Myalgia	1.7	1.5
Disorders			Psychiatric Disorders		
Headache	20.3	19.0	Insomnia	16.4	8.8
Dizziness	11.7	6.7	Sexual Dysfunction-Male (1)	15.5	2.2
Tremor	10.7	2.7	Somnolence	13.4	5.9
Paresthesia	2.0	1.8	Agitation	5.6	4.0
Hypoesthesia	1.7	0.6	Nervousness	3.4	1.9
Twitching	1.4	0.1	Anxiety	2.6	1.3
Hypomania	1.3	0.4	Yawning	1.9	0.2
Disorders of Skin and Appendages			Sexual Dysfunction-Female (2)	1.7	0.2
Rash	2.1	1.5	Concentration Impaired	1.3	0.5
Gastrointestinal Disorders			Reproductive		
Nausea	26.1	11.8	Menstrual Disorder (2)	1.0	0.5
Diarrhea/Loose Stools	17.7	9.3	Respiratory System Disorders		
Constipation	8.4	6.3	Rhinitis	2.0	1.5
Dyspepsia	6.0	2.8	Pharyngitis	1.2	0.9
Vomiting	3.8	1.8	Special Senses		
Flatulence	3.3	2.5	Vision Abnormal	4.2	2.1
Anorexia	2.8	1.6	Tinnitus	1.4	1.1
Abdominal Pain	2.4	2.2	Taste Perversion	1.2	0.7
Appetite Increased	1.3	0.9	Urinary System Disorders		
General			Micturition Frequency	2.0	1.2
Fatigue	10.6	8.1	Micturition Disorder	1.4	0.5

*Events reported by at least 1% of patients treated with ZOLOFT are included.

(1) - % based on male patients only; 271 ZOLOFT (primarily ejaculatory delay) and 271 placebo patients.

(2) - % based on female patients only; 590 ZOLOFT and 582 placebo patients.

Other Events Observed During the Premarketing Evaluation of ZOLOFT (sertraline hydrochloride): During its premarketing assessment, multiple doses of ZOLOFT were administered to approximately 2700 subjects. Events are further categorized by body system and listed in order of decreasing frequency according to the following definitions: frequent adverse events are those occurring on one or more occasions in at least 1/100 patients (only those not already listed in the tabulated results from placebo-controlled trials appear in this listing); infrequent adverse events are those occurring in 1/100 to 1/1000 patients; rare events are those occurring in fewer than 1/1000 patients. Events of major clinical importance are also described in the PRECAUTIONS section. **Autonomic Nervous System Disorders—Infrequent:** flushing, mydriasis, increased salivary, cold clammy skin; **Rare:** pallor. **Cardiovascular—Infrequent:** postural dizziness, hypertension, hypotension, postural hypotension, edema, dependent edema, periorbital edema, peripheral edema, peripheral ischemia, syncope, tachycardia; **Rare:** precordial chest pain, substernal chest pain, aggravated hypertension, myocardial infarction, varicose veins. **Control and Peripheral Nervous System Disorders—Frequent:** confusion; **Infrequent:** ataxia, abnormal coordination, abnormal gait, hyperesthesia, hyperkinesia, hypokinesia, migraine, nystagmus, vertigo; **Rare:** local anesthesia, coma, convulsions, dyskinesia, dysphasia, hyperreflexia, hypotonia, ptosis. **Disorders of Skin and Appendages—Infrequent:** acne, alopecia, pruritus, erythematous rash, maculopapular rash, dry skin; **Rare:** bullous eruption, dermatitis, erythema multiforme, abnormal hair texture, hypertrichosis, photosensitivity reaction, follicular rash, skin discoloration, abnormal skin odor, urticaria. **Endocrine Disorders—Rare:** exophthalmos, gynecomastia. **Gastrointestinal Disorders—Infrequent:** dysphagia, eructation; **Rare:** diverticulitis, fecal incontinence, gastritis, gastroenteritis, glossitis, gum hyperplasia, hemorrhoids, hiccup, melena, hemorrhagic peptic ulcer, proctitis, stomatitis, ulcerative stomatitis, tenesmus, tongue edema, tongue ulceration. **General—Frequent:** asthenia; **Infrequent:** malaise, generalized edema, rigors, weight decrease, weight increase; **Rare:** enlarged abdomen, halitosis, otitis media, aphthous stomatitis. **Hematopoietic and Lymphatic—Infrequent:** lymphadenopathy, purpura; **Rare:** anemia, anterior chamber eye hemorrhage. **Metabolic and Nutritional Disorders—Rare:** dehydration, hypercholesterolemia, hypoglycemia. **Musculoskeletal System Disorders—Infrequent:** arthralgia, arthrosis, dystonia, muscle cramps, muscle weakness; **Rare:** hernia. **Psychiatric Disorders—Infrequent:** abnormal dreams, aggressive reaction, amnesia, opacity, delusion, depersonalization, depression, aggravated depression, emotional lability, euphoria, hallucination, neurosis, paranoid reaction, suicide ideation and attempt, teeth-grinding, abnormal thinking; **Rare:** hysteria, somnambulism, withdrawal syndrome. **Reproductive—Infrequent:** dysmenorrhea (2), intermenstrual bleeding (2); **Rare:** amenorrhea (2), balanoposthitis (1), breast enlargement (2), female breast pain (2), leukorrhea (2), menorrhagia (2), atrophic vaginitis (2).

(1) - % based on male subjects only: 1005; (2) - % based on female subjects only: 1705.

Respiratory System Disorders—Infrequent: bronchospasm, coughing, dyspnea, epistaxis; **Rare:** bradypnea, hyperventilation, sinusitis, stridor. **Special Senses—Infrequent:** abnormal accommodation, conjunctivitis, diplopia, earache, eye pain, xerophthalmia; **Rare:** abnormal lacrimation, photophobia, visual field defect. **Urinary System Disorders—Infrequent:** dysuria, face edema, nocturia, polyuria, urinary incontinence; **Rare:** oliguria, renal pain, urinary retention. **Laboratory Tests:** In man, asymptomatic elevations in serum transaminases (SGOT [or AST] and SGPT [or ALT]) have been reported infrequently (approximately 0.8%) in association with ZOLOFT administration. These hepatic enzyme elevations usually occurred within the first 1 to 9 weeks of drug treatment and promptly diminished upon drug discontinuation. ZOLOFT therapy was associated with small mean increases in total cholesterol (approximately 3%) and triglycerides (approximately 5%), and a small mean decrease in serum uric acid (approximately 7%) of no apparent clinical importance. **DRUG ABUSE AND DEPENDENCE Controlled Substance Class -** ZOLOFT (sertraline hydrochloride) is not a controlled substance. **Physical and Psychological Dependence -** ZOLOFT has not been systematically studied, in animals or humans, for its potential for abuse, tolerance, or physical dependence. However, the premarketing clinical experience with ZOLOFT did not reveal any tendency for a withdrawal syndrome or any drug-seeking behavior. As with any new CNS active drug, physicians should carefully evaluate patients for history of drug abuse and follow such patients closely, observing them for signs of ZOLOFT misuse or abuse (e.g., development of tolerance, incrementation of dose, drug-seeking behavior). **OVERDOSAGE Human Experience -** There have been 3 cases of ZOLOFT (sertraline hydrochloride) overdosage (approximately 750-2,100 mg). No specific therapy was required for any of the 3 patients, all of whom recovered completely. **Management of Overdosage -** Establish and maintain an airway, insure adequate oxygenation and ventilation. Activated charcoal, which may be used with sorbitol, may be as or more effective than emesis or lavage, and should be considered in treating overdosage. Cardiac and vital signs monitoring is recommended along with general symptomatic and supportive measures. There are no specific antidotes for ZOLOFT. Due to the large volume of distribution of ZOLOFT, forced diuresis, dialysis, hemoperfusion, and exchange transfusion are unlikely to be of benefit. In managing overdosage, consider the possibility of multiple drug involvement. The physician should consider contacting a poison control center on the treatment of any overdosage.



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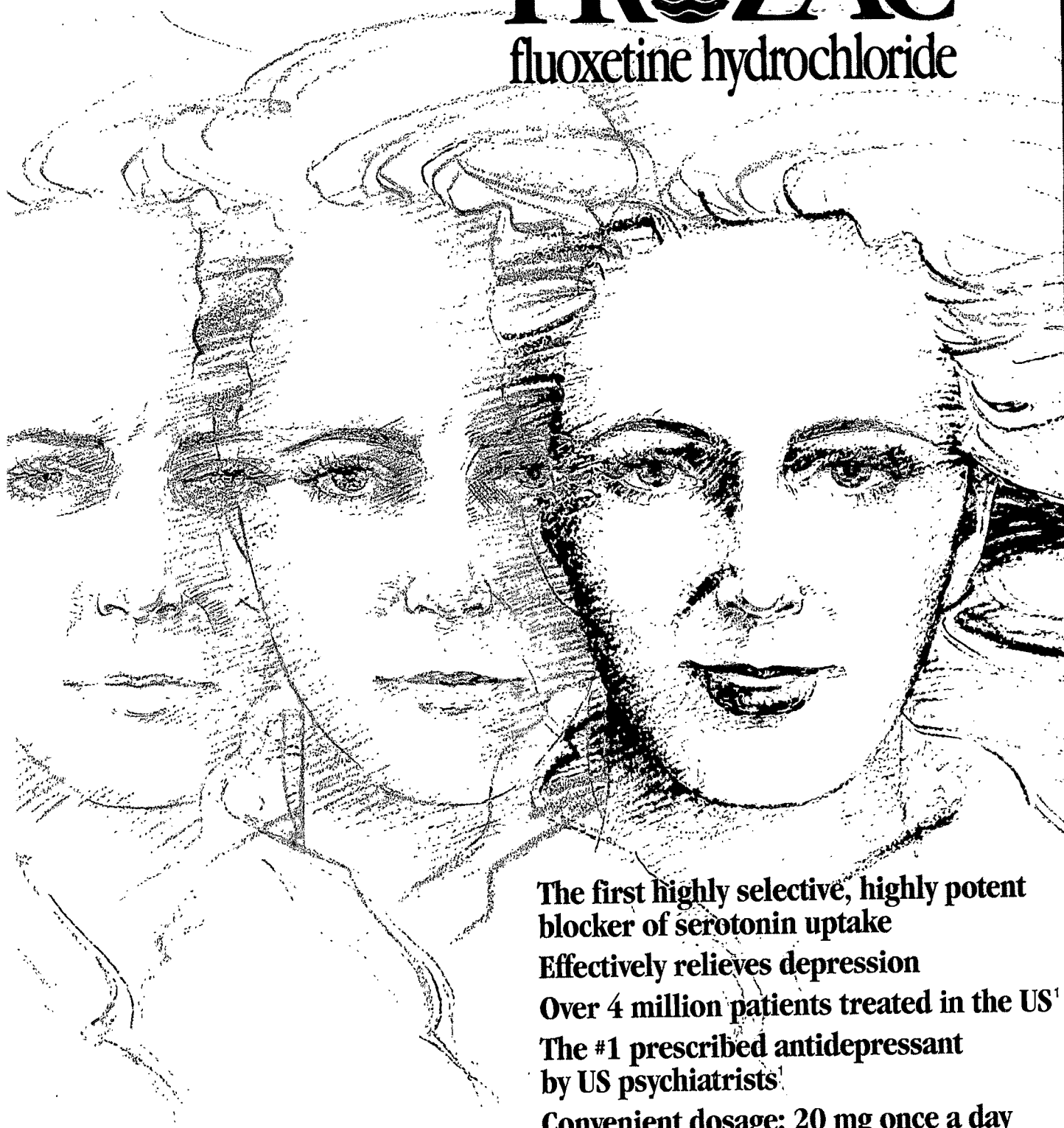
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Contraindications: Known hypersensitivity to Prozac.

Monamine Oxidase Inhibitors — There have been reports of serious, sometimes fatal, reactions in patients receiving fluoxetine in combination with an MAOI and in patients who have recently discontinued fluoxetine and are then started on an MAOI. Some cases presented with features resembling neuroleptic malignant syndrome.

Wait at least 14 days between discontinuing an MAOI and starting therapy with Prozac. Because of the long half-lives of fluoxetine and its active metabolite, wait at least 5 weeks between discontinuing Prozac and starting therapy with an MAOI. Prozac should not be used concomitantly with MAOIs.

Warnings: Rash and Possibly Allergic Events — Approximately 4% of 5,600 fluoxetine patients developed a rash and/or urticaria in premarketing testing. Almost a third of these discontinued therapy because of rash and/or associated systemic signs or symptoms. Reported in association with rash were fever, leukocytosis, arthralgias, edema, carpal tunnel syndrome, respiratory distress, lymphadenopathy, proleluria, and mild transaminase elevation. Most patients improved promptly upon discontinuation of fluoxetine and/or adjunctive treatment with antihistamines or steroids, and all were reported to recover completely.

Of 2 patients who developed a serious cutaneous systemic illness during premarketing clinical trials, 1 was considered to have a leukocytoclastic vasculitis, and the other, a severe desquamating syndrome considered variously to be a vasculitis or erythema multiforme. Other patients have had systemic syndromes suggestive of serum sickness. Since the introduction of Prozac, systemic events possibly related to vasculitis have developed in patients with rash. Although these events are rare, they may be serious, involving the lung, kidney, or liver. Death has been reported to occur in association with these systemic events.

Anaphylactoid events, including bronchospasm, angioedema, and urticaria alone and in combination, have been reported.

Pulmonary events, including inflammatory processes of varying histopathology and/or fibrosis, have been reported rarely. These events have occurred with dyspnea as the only preceding symptom.

Whether these systemic events and rash have a common underlying cause or represent immunologic responses is not known. Upon the appearance of rash or of other possibly allergic phenomena for which an alternative etiology cannot be identified, Prozac should be discontinued.

Precautions: General — **Anxiety, Nervousness, and Insomnia** — Reported by 10% to 15% of patients, 5% of whom discontinued fluoxetine.

Altered Appetite and Weight — Significant weight loss, especially in underweight patients, may be an undesirable result of treatment.

Approximately 9% of fluoxetine patients experienced anorexia in controlled clinical trials, an incidence approximately sixfold that seen with placebo. A weight loss >5% of body weight occurred in 13% of fluoxetine patients compared with 4% in those on placebo and 3% in those on tricyclics. However, only rarely did fluoxetine patients discontinue treatment because of weight loss.

Activation of Mania/Hypomania — Hypomania or mania occurred in approximately 1% of fluoxetine patients in premarketing testing.

Seizures — Twelve of 6,000 patients (0.2%) experienced convulsions (or, possibly, seizures). Prozac should be introduced with care in patients with a history of seizures.

Suicide — Close supervision of high-risk patients should accompany initial therapy. Prescriptions of Prozac should be written for the smallest quantity of capsules consistent with good patient management to reduce the risk of overdose.

The Long Elimination Half-Lives of Fluoxetine and Its Metabolites — Because of the long elimination half-lives of the parent drug (2 to 3 days) and its major active metabolite (7 to 9 days), changes in dose will not be fully reflected in plasma for several weeks, affecting both strategies for titration to final dose and withdrawal from treatment.

Use in Patients with Concomitant Illness — Caution is advisable in patients with diseases or conditions that could affect metabolism or hemodynamic responses.

Fluoxetine has not been evaluated or used to any appreciable extent in patients with a recent history of myocardial infarction or unstable heart disease. ECGs of 312 patients who received Prozac in double-blind trials were retrospectively evaluated; no conduction abnormalities that resulted in heart block were observed. The mean heart rate was reduced by approximately 3 beats/min.

In subjects with cirrhosis, the clearances of fluoxetine and its active metabolite were decreased. A lower or less frequent dose should be used in patients with cirrhosis.

Fluoxetine should be used with caution in patients with severe renal impairment.

In patients with diabetes, Prozac may alter glycemic control. Hypoglycemia has occurred during therapy with Prozac, and hyperglycemia has developed following discontinuation. Insulin and/or oral hypoglycemic dosage may need to be adjusted when fluoxetine therapy is instituted or discontinued.

Interference with Cognitive and Motor Performance — Any psychoactive drug may impair judgment, thinking, or motor skills, and patients should be cautioned about operating hazardous machinery, including automobiles, until they are reasonably certain that the drug does not affect them adversely.

Information for Patients — Physicians should advise their patients to notify them if they:

- are taking or plan to take any prescription or over-the-counter drugs or alcohol
- become pregnant or intend to become pregnant during therapy
- are breast feeding an infant
- develop a rash or hives

Drug Interactions — **Tryptophan** — Five patients receiving tryptophan experienced adverse reactions, including agitation, restlessness, and gastrointestinal distress.

Monamine Oxidase Inhibitors — See Contraindications.

Other Antidepressants — There have been greater than 2-fold increases of previously stable plasma levels of other antidepressants when Prozac has been administered in combination with these agents.

Lithium — There have been reports of both increased and decreased lithium levels and lithium toxicity. Lithium levels should be monitored.

Diazepam Clearance — The half-life of diazepam may be prolonged in some patients.

Drugs Tightly Bound to Plasma Proteins — Because fluoxetine is tightly bound to plasma protein, the concurrent administration of fluoxetine and another tightly bound drug may cause a shift in plasma concentrations potentially resulting in an adverse effect. Adverse effects may also result from displacement of protein-bound fluoxetine by other tightly bound drugs.

CNS-Active Drugs — Caution is advised if the concomitant administration of Prozac and such drugs is required.

Electroconvulsive Therapy — There have been rare reports of prolonged seizures in patients on fluoxetine receiving ECT treatment.

Carcinogenesis, Mutagenesis, Impairment of Fertility — There is no evidence of carcinogenicity, mutagenicity, or impairment of fertility with Prozac.

The dietary administration of fluoxetine to rats and mice for 2 years at doses approximately 7.5 and 9.0 times the maximum human dose (80 mg) respectively revealed no evidence of carcinogenicity.

Fluoxetine and norfluoxetine have been shown to have no genotoxic effects based on the following assays: bacterial mutation assay, DNA repair assay in cultured rat hepatocytes, mouse lymphoma assay, and in vivo sister chromatid exchange assay in Chinese hamster bone marrow cells.

Two fertility studies in rats at doses approximately 5 and 9 times the maximum human dose (80 mg) respectively revealed no adverse effects on fertility. A slight decrease in neonatal survival was noted, probably associated with depressed maternal food consumption and suppressed weight gain.

Pregnancy — **Teratogenic Effects** — **Pregnancy Category B** — Reproduction studies in rats and rabbits at doses 9 and 11 times the maximum human dose (80 mg) respectively revealed no evidence of harm to the fetus. Although there have been no adequate and well-controlled studies in pregnant women, this drug should be used during pregnancy only if clearly needed.

Labor and Delivery — The effect of Prozac on labor and delivery in humans is unknown.

Nursing Mothers — Because Prozac is known to be excreted in human milk, caution should be exercised when Prozac is administered to a nursing woman.

Use in Children — Safety and effectiveness in children have not been established.

Use in the Elderly — In clinical studies of several hundred elderly patients, no unusual adverse age-related phenomena were identified. However, these data are insufficient to rule out possible age-related differences during chronic use, particularly in elderly patients with concomitant systemic illnesses or those receiving concomitant drugs.

Hyponatremia — Hyponatremia (some cases with serum Na <110 mmol/L) has been reported, which appeared to be reversible on drug discontinuation. Some cases were possibly due to SIADH, and the majority have been in older patients and those taking diuretics or otherwise volume depleted.

Platelet Function — There have been rare reports of altered platelet function and/or abnormal results from laboratory studies in patients taking fluoxetine. While there have been reports of abnormal bleeding in several patients taking fluoxetine, it is unclear whether fluoxetine had a causative role.

Adverse Reactions: Commonly Observed — Nervous system complaints, including anxiety, nervousness, and insomnia; drowsiness and fatigue or asthenia; tremor; sweating; gastrointestinal complaints, including anorexia, nausea, and diarrhea; and dizziness or lightheadedness.

Associated With Discontinuation of Treatment — Fifteen percent of 4,000 clinical trial patients discontinued fluoxetine due to an adverse event. The more common events included: psychiatric (5.3%), primarily nervousness, anxiety, and insomnia; digestive (3.0%), primarily nausea; nervous system (1.6%), primarily dizziness; body as a whole (1.5%), primarily asthenia and headache; and skin (1.4%), primarily rash and pruritus.

Incidence in Controlled Clinical Trials — The accompanying table enumerates adverse events that occurred at a frequency of $\geq 1\%$ in controlled trials.

Other Events Observed During Premarketing Evaluation in 5,600 Fluoxetine Patients — Frequent adverse events are defined as those occurring on 1 or more occasions in at least 1/100 patients; infrequent adverse events are those occurring in 1/100 to 1/1,000 patients; rare events are those occurring in less than 1/1,000 patients.

Body as a Whole — **Frequent:** chills; **Infrequent:** chills and fever, cyst, face edema, hanger effect, jaw pain, malaise, neck pain, neck rigidity, and pelvic pain; **Rare:** abdomen enlarged, cellulitis, hydrocephalus, hypothermia, LE syndrome, moniliasis, and serum sickness.

Cardiovascular System — **Infrequent:** angina pectoris, arrhythmia, hemorrhage, hypertension, hypotension, migraine, postural hypotension, syncope, and tachycardia; **Rare:** AV block first-degree, bradycardia, bundle branch block, cerebral ischemia, myocardial infarct, thrombophlebitis, vascular headache, and ventricular arrhythmia.

Digestive System — **Frequent:** increased appetite; **Infrequent:** aphthous stomatitis, dysphagia, eructation, esophagitis, gastritis, gingivitis, glossitis, liver function tests abnormal, melena, stomatitis, thirst; **Rare:** bloody diarrhea, cholecystitis, cholelithiasis, colitis, duodenal ulcer, enteritis, fecal incontinence, hematemesis, hepatitis, hepatomegaly, hyperchlorhydria, increased salivation, jaundice, liver tenderness, mouth ulceration, salivary gland enlargement, stomach ulcer, tongue discoloration, and tongue edema.

Endocrine System — **Infrequent:** hypothyroidism; **Rare:** goiter and hyperthyroidism.

Hemic and Lymphatic System — **Infrequent:** anemia and lymphadenopathy; **Rare:** bleeding time increased, blood dyscrasia, leukopenia, lymphocytosis, ptechia, purpura, sedimentation rate increased, and thrombocytopenia.

Metabolic and Nutritional — **Frequent:** weight loss; **Infrequent:** generalized edema, hypoglycemia, peripheral edema, and weight gain; **Rare:** dehydration, gout, hypercholesterolemia, hyperglycemia, hyperlipemia, hypoglycemic reaction, hypokalemia, hyponatremia, and iron deficiency anemia.

Musculoskeletal System — **Infrequent:** arthritis, bone pain, bursitis, tenosynovitis, and twitching; **Rare:** bone necrosis, chondrodystrophy, muscle hemorrhage, myositis, osteoporosis, pathological fracture, and rheumatoid arthritis.

Nervous System — **Frequent:** abnormal dreams and agitation; **Infrequent:** abnormal gait, acute brain syndrome, akathisia, amnesia,

TREATMENT-EMERGENT ADVERSE EXPERIENCE INCIDENCE IN PLACEBO-CONTROLLED CLINICAL TRIALS

Body System/ Adverse Event*	Prozac (N = 1,730)	Placebo (N = 799)	Body System/ Adverse Event*	Prozac (N = 1,730)	Placebo (N = 799)
Nervous			Body as a Whole		
Headache	20.3	15.5	Asymptomatic	4.4	1.9
Nervousness	14.9	8.5	Infection, viral	3.4	3.1
Insomnia	13.8	7.1	Pain, limb	1.6	1.1
Drowsiness	11.6	8.3	Fever	1.4	—
Anxiety	9.4	5.5	Pain, chest	1.3	1.1
Tremor	7.9	2.4	Allergy	1.2	1.1
Dizziness	5.7	3.3	Influenza	1.2	1.5
Fatigue	4.2	1.1	Respiratory		
Sedated	1.9	1.3	Upper respiratory infection	7.6	6.0
Sensation disturbance	1.7	2.0	Flu-like syndrome	2.8	1.9
Lbido, decreased	1.6	—	Pharyngitis	2.7	1.3
Light-headedness	1.6	—	Nasal congestion	2.6	2.3
Concentration, decreased	1.5	—	Headache, sinus	2.3	1.8
Digestive			Sinusitis	2.1	2.0
Nausea	21.1	10.1	Cough	1.6	1.6
Diarrhea	12.3	7.0	Dyspnea	1.4	—
Mouth dryness	9.5	6.0	Cardiovascular		
Anorexia	8.7	1.5	Hot flashes	1.8	1.0
Dyspepsia	6.4	4.3	Palpitations	1.3	1.4
Constipation	4.5	3.3	Musculoskeletal		
Pain, abdominal	3.4	2.9	Pain, back	2.0	2.4
Vomiting	2.4	1.3	Pain, joint	1.2	1.1
Taste change	1.8	—	Pain, muscle	1.2	1.0
Fatulence	1.6	1.1	Urogenital		
Gastroenteritis	1.0	1.4	Urinary tract infection	1.2	—
Skin and Appendages			Special Senses		
Sweating, excessive	8.4	3.8	Visual disturbance	2.8	1.8
Rash	2.7	1.8			
Pruritus	2.4	1.4			

*Events reported by $\geq 1\%$ of fluoxetine patients are included.
— Incidence <1%.

apathy, ataxia, buccoglossal syndrome, CNS stimulation, convulsion, delusions, depersonalization, emotional lability, euphoria, hallucinations, hostility, hyperkinesia, hypesthesia, incoordination, libido increased, manic reaction, neuralgia, neuropathy, paranoid reaction, psychosis, and vertigo; **Rare:** abnormal electroencephalogram, amniotic reaction, chronic brain syndrome, circumoral paresthesia, CNS depression, coma, dysarthria, dystonia, extrapyramidal syndrome, hypertonia, hysteria, myoclonus, nystagmus, paralysis, reflexes decreased, stupor, and torticollis.

Respiratory System — **Frequent:** bronchitis, rhinitis, and yawn; **Infrequent:** asthma, epistaxis, hiccup, hyperventilation, and pneumonia; **Rare:** apnea, hemoptysis, hypoxia, larynx edema, lung edema, lung fibrosis/alveolitis, and pleural effusion.

Skin and Appendages — **Infrequent:** acne, alopecia, contact dermatitis, dry skin, herpes simplex, maculopapular rash, and urticaria; **Rare:** eczema, erythema multiforme, fungal dermatitis, herpes zoster, hirsutism, psoriasis, purpuric rash, pustular rash, seborrhea, skin discoloration, skin hypertrophy, subcutaneous nodule, and vesiculobullous rash.

Special Senses — **Infrequent:** amblyopia, conjunctivitis, ear pain, eye pain, mydriasis, photophobia, and tinnitus; **Rare:** blepharitis, cataract, corneal lesion, deafness, diplopia, eye hemorrhage, glaucoma, iritis, ptosis, strabismus, and taste loss.

Urogenital System — **Infrequent:** abnormal ejaculation, amenorrhea, breast pain, cystitis, dysuria, fibrocystic breast, impotence, leukorrhea, menopause, menorrhagia, ovarian disorder, urinary incontinence, urinary retention, urinary urgency, urination impaired, and vaginitis; **Rare:** abortion, albuminuria, breast enlargement, dyspareunia, epididymitis, female lactation, hematuria, hypomenorrhea, kidney calculus, metrorrhagia, orchitis, polyuria, pyelonephritis, pyuria, salpingitis, urethral pain, urethritis, urinary tract disorder, urinary lithiasis, uterine hemorrhage, uterine spasm, and vaginal hemorrhage.

Postintroduction Reports — Voluntary reports of adverse events temporally associated with Prozac that have been received since market introduction and which may have no causal relationship with the drug include the following: aplastic anemia, cerebral vascular accident, confusion, dyskinesia (including, for example, a case of buccal-lingual-masticatory syndrome with involuntary tongue protrusion reported to develop in a 77-year old female after 5 weeks of fluoxetine therapy and which completely resolved over the next few months following drug discontinuation), echymoses, eosinophilic pneumonia, gastrointestinal hemorrhage, hyperprolactinemia, immune-related hemolytic anemia, movement disorders developing in patients with risk factors including drugs associated with such events and worsening of preexisting movement disorders, neuroleptic malignant syndrome-like events, pancreatitis, pancytopenia, suicidal ideation, thrombocytopenia, thrombocytopenic purpura, vaginal bleeding after drug withdrawal, and violent behaviors.

Overdosage: Human Experience — As of December 1987, there were 2 deaths among approximately 38 reports of acute overdose with fluoxetine, either alone or in combination with other drugs and/or alcohol. One death involved a combined overdose with approximately 1,800 mg of fluoxetine and an undetermined amount of meprobamate. A second death involved fluoxetine, codeine, and temazepam.

One other patient who reportedly took 3,000 mg of fluoxetine experienced 2 grand mal seizures that remitted spontaneously without specific anticonvulsant treatment. The actual amount of drug absorbed may have been less due to vomiting.

Nausea and vomiting were prominent in overdoses involving higher fluoxetine doses. Other prominent symptoms of overdose included agitation, restlessness, hypomania, and other signs of CNS excitation. Except for the 2 deaths noted above, all other overdose cases recovered without residual.

Since introduction, reports of death attributed to overdosage of fluoxetine alone have been extremely rare.

PV 2475 DPP

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Additional information available to the profession upon request.



Dista Products Company
Division of Eli Lilly and Company
Indianapolis, Indiana 46285

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Prozac® (fluoxetine hydrochloride)

Prozac® (fluoxetine hydrochloride)

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**Why are more
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Impressive results!

WELLBUTRIN is as effective as Prozac®** in relieving depression. In a 6-week, double-blind study of 123 depressed outpatients, WELLBUTRIN was as effective as Prozac, with a comparable side effects profile.¹

WELLBUTRIN has demonstrated therapeutic efficacy comparable to tricyclics,^{2,3} with fewer of the tricyclic-associated side effects that can disrupt therapy.³⁻⁷

In the non-responder: 54% of 1,301 patients who responded poorly to their previous antidepressant were reported to have had a good response to WELLBUTRIN in a surveillance study† of 3,341 patients.⁸

In the non-tolerator: 63% of 766 patients who poorly tolerated their previous antidepressant treatment were reported in the same surveillance study† to have had good tolerance of WELLBUTRIN.⁸

High rates of compliance^{1,8} and flexible dosing with 75 mg and 100 mg tablets.

Efficacy demonstrated in both inpatients^{4,9} and outpatients.¹⁰



Unique, non-serotonergic

Wellbutrin®
(BUPROPION HCl)

Tablets 75 mg and 100 mg

In a class by itself.

The principal medically important adverse reaction with WELLBUTRIN is seizure, which occurs in approximately one-tenth of one percent (4 out of 1000) of patients. This incidence may exceed that of other marketed antidepressants although no direct comparative studies have been conducted. For more information about dosing to achieve optimal patient response and recommendations for reducing the risk of side effects, see full prescribing information, especially the DOSAGE AND ADMINISTRATION and WARNINGS sections.

See brief summary of full prescribing information on last page of this advertisement.

*Prozac (fluoxetine HCl) is a registered trademark of Dista Products Co., a division of Eli Lilly and Company.

†Mean daily dose at week 6 was 382 mg for WELLBUTRIN patients and 38 mg for fluoxetine patients.

‡In a 3,341-patient surveillance study designed to determine the incidence of seizures with bupropion under conditions of general clinical practice, physicians were asked to report whether patients' responses to and tolerance of the antidepressant received in the month before the study were good, poor, or unknown. In the month before the study, 1,902 patients received previous antidepressant treatment. The results reported reflected evaluations of response to and tolerance of WELLBUTRIN at the end of up to 56 days of treatment.⁸

WELLBUTRIN® (BUPROPION HYDROCHLORIDE) Tablets

Before prescribing, please consult complete product information, a summary of which follows:
INDICATIONS AND USAGE: Wellbutrin is indicated for the treatment of depression. A physician considering the initiation of Wellbutrin should be aware that the drug may cause generalized seizures with an approximate incidence of 0.4% (4/1000). This incidence may exceed that of other antidepressants as much as fourfold. This relative risk is only an approximation since no direct comparative studies have been conducted.
CONTRAINDICATIONS: Wellbutrin is contraindicated in patients: with a seizure disorder; with a current or prior diagnosis of bulimia or anorexia nervosa, because of a higher incidence of seizures noted in such patients; who have shown an allergic response to it; or who are currently being treated with an MAO inhibitor. At least 14 days should elapse between discontinuation of an MAO inhibitor and initiation of treatment with Wellbutrin.

WARNINGS: SEIZURES: Wellbutrin is associated with seizures in approximately 0.4% (4/1000) of patients treated at doses up to 450 mg/day. This incidence of seizures may exceed that of other marketed antidepressants by as much as fourfold. This relative risk is only an approximate estimate because no direct comparative studies have been conducted. The estimated seizure incidence for Wellbutrin increases almost tenfold between 450 and 600 mg/day, which is twice the usually required daily dose (300 mg) and one and one-third the maximum recommended daily dose (450 mg). Given the wide variability among individuals and their capacity to metabolize and eliminate drugs, this disproportionate increase in seizure incidence with dose incrementation calls for caution in dosing.
 During the initial development, 25 among approximately 2400 patients treated with Wellbutrin experienced seizures. At the time of seizure, 7 patients were receiving daily doses of 450 mg or below, for an incidence of 0.33% (3/1000) within the recommended dose range. Twelve (12) patients experienced seizures at 600 mg per day (2.3% incidence); 6 additional patients had seizures at daily doses between 600 and 900 mg (2.8% incidence).

A separate, prospective study was conducted to determine the incidence of seizure during an 8 week treatment exposure in approximately 3200 additional patients who received daily doses of up to 450 mg. Patients were permitted to continue treatment beyond 8 weeks if clinically indicated. Eight (8) seizures occurred during the initial 8 week treatment period and 5 seizures were reported in patients continuing treatment beyond 8 weeks, resulting in a total seizure incidence of 0.4%. The risk of seizure appears to be strongly associated with dose and the presence of predisposing factors. A significant predisposing factor (e.g., history of head trauma or prior seizure, CNS tumor, concomitant medications that lower seizure threshold, etc.) was present in approximately one-half of the patients experiencing a seizure. Sudden and large increments in dose may contribute to increased risk. While many seizures occurred early in the course of treatment, some seizures did occur after several weeks at fixed dose.

Recommendations for reducing the risk of seizure: Retrospective analysis of clinical experience gained during the development of Wellbutrin suggests that the risk of seizure may be minimized if (1) the total daily dose of Wellbutrin does not exceed 450 mg, (2) the daily dose is administered t.i.d., with each single dose not to exceed 150 mg to avoid high peak concentrations of bupropion and/or its metabolites, and (3) the rate of incrementation of dose is very gradual. Extreme caution should be used when Wellbutrin is (1) administered to patients with a history of seizure, cranial trauma, or other predisposition(s) toward seizure, or (2) prescribed with other agents (e.g., antipsychotics, other antidepressants, etc.) or treatment regimens (e.g., abrupt discontinuation of a benzodiazepine) that lower seizure threshold.

Potential for Hepatotoxicity: In rats receiving large doses of bupropion chronically, there was an increase in incidence of hepatic hyperplastic nodules and hepatocellular hypertrophy. In dogs receiving large doses of bupropion chronically, various histologic changes were seen in the liver, and laboratory tests suggesting mild hepatocellular injury were noted. Although scattered abnormalities in liver function tests were detected in patients participating in clinical trials, there is no clinical evidence that bupropion acts as a hepatotoxin in humans.

PRECAUTIONS: General:

Agitation and Insomnia: A substantial proportion of patients treated with Wellbutrin experience some degree of increased restlessness, agitation, anxiety, and insomnia, especially shortly after initiation of treatment. In clinical studies, these symptoms were sometimes of sufficient magnitude to require treatment with sedative/hypnotic drugs. In approximately 2% of patients, symptoms were sufficiently severe to require discontinuation of Wellbutrin treatment.

Psychosis, Confusion, and Other Neuropsychiatric Phenomena: Patients treated with Wellbutrin have been reported to show a variety of neuropsychiatric signs and symptoms including delusions, hallucinations, psychotic episodes, confusion, and paranoia. Because of the uncontrolled nature of many studies, it is impossible to provide a precise estimate of the extent of risk imposed by treatment with Wellbutrin. In several cases, neuropsychiatric phenomena abated upon dose reduction and/or withdrawal of treatment.

Activation of Psychosis and/or Mania: Antidepressants can precipitate manic episodes in Bipolar Manic Depressive patients during the depressed phase of their illness and may activate latent psychosis in other susceptible patients. Wellbutrin is expected to pose similar risks.

Altered Appetite and Weight: A weight loss of greater than 5 pounds occurred in 28% of Wellbutrin patients. This incidence is approximately double that seen in comparable patients treated with tricyclics or placebo. Furthermore, while 34.5% of patients receiving tricyclic antidepressants gained weight, only 9.4% of patients treated with Wellbutrin did. Consequently, if weight loss is a major presenting sign of a patient's depressive illness, the anorectic and/or weight reducing potential of Wellbutrin should be considered.

Suicide: The possibility of a suicide attempt is inherent in depression and may persist until significant remission occurs. Accordingly, prescriptions for Wellbutrin should be written for the smallest number of tablets consistent with good patient management.

Use in Patients with Systemic Illness: There is no clinical experience establishing the safety of Wellbutrin in patients with a recent history of myocardial infarction or unstable heart disease. Therefore, care should be exercised if it is used in these groups.

Because bupropion HCl and its metabolites are almost completely excreted through the kidney and metabolites are likely to undergo conjugation in the liver prior to urinary excretion, treatment of patients with renal or hepatic impairment should be initiated at reduced dosage as bupropion and its metabolites may accumulate in such patients beyond concentrations expected in patients without renal or hepatic impairment. The patient should be closely monitored for possible toxic effects of elevated blood and tissue levels of drug and metabolites. Information for Patients: Consult complete product information.

Drug Interactions: No systematic data have been collected on the consequences of the concomitant administration of Wellbutrin and other drugs.

However, animal data suggest that Wellbutrin may be an inducer of drug metabolizing enzymes. This may be of potential clinical importance because the blood levels of co-administered drugs may be altered. Alternatively, because bupropion is extensively metabolized, the co-administration of other drugs may affect its clinical activity. In particular, care should be exercised when administering drugs known to affect hepatic drug metabolizing enzyme systems (e.g., carbamazepine, cimetidine, phenobarbital, phenytoin). Studies in animals demonstrate that the acute toxicity of bupropion is enhanced by the MAO inhibitor phenelzine (see CONTRAINDICATIONS).

Limited clinical data suggest a higher incidence of adverse experiences in patients receiving concurrent administration of Wellbutrin and L-dopa. Administration of Wellbutrin to patients receiving L-dopa concurrently should be undertaken with caution, using small initial doses and small gradual dose increases. Concurrent administration of Wellbutrin and agents which lower seizure threshold should be undertaken only with extreme caution (see WARNINGS). Low initial dosing and small gradual dose increases should be employed.
Carcinogenesis, Mutagenesis, Impairment of Fertility: Lifetime carcinogenicity studies were performed in rats and mice at doses up to 300 and 150 mg/kg/day, respectively. In the rat study there was an increase in nodular proliferative lesions of the liver at doses of 100 to 300 mg/kg/day; lower doses were not tested. The question of whether or not such lesions may be precursors of neoplasms of the liver is currently unresolved. Similar liver lesions were not seen in the mouse study, and no increase in malignant tumors of the liver and other organs was seen in either study.

Bupropion produced a borderline positive response (2-3 times control mutation rate) in some strains in the Ames bacterial mutagenicity test, and a high oral dose (300, but not 100 or 200 mg/kg) produced a low incidence of chromosomal aberrations in rats. The relevance of these results in estimating the risk of human exposure to therapeutic doses is unknown.

A fertility study was performed in rats; no evidence of impairment of fertility was encountered at oral doses up to 300 mg/kg/day.

Pregnancy: Teratogenic Effects: Pregnancy Category B: Reproduction studies have been performed in rabbits and rats at doses up to 15-45 times the human daily dose and have revealed no definitive evidence of impaired fertility or harm to the fetus due to bupropion. (In rabbits, a slightly increased incidence of fetal abnormalities was seen in two studies, but there was no increase in any specific abnormality). There are no adequate and well-controlled studies in pregnant women. Because animal reproduction studies are not always predictive of human response, this drug should be used during pregnancy only if clearly needed.

Labor and Delivery: The effect of Wellbutrin on labor and delivery in humans is unknown.

Nursing Mothers: Because of the potential for serious adverse reactions in nursing infants from Wellbutrin, a decision should be made whether to discontinue nursing or to discontinue the drug, taking into account the importance of the drug to the mother.

Pediatric Use: The safety and effectiveness of Wellbutrin in individuals under 18 years old have not been established.

Use in the Elderly: Wellbutrin has not been systematically evaluated in older patients.

ADVERSE REACTIONS: (See also WARNINGS and PRECAUTIONS) Adverse events commonly encountered in patients treated with Wellbutrin are agitation, dry mouth, insomnia, headache/migraine, nausea/vomiting constipation, and tremor.

Adverse events were sufficiently troublesome to cause discontinuation of Wellbutrin treatment in approximately ten percent of the 2400 patients and volunteers who participated in clinical trials during the product's initial development. The more common events causing discontinuation include neuropsychiatric disturbances (3.0%), primarily agitation and abnormalities in mental status; gastrointestinal disturbances (2.1%), primarily nausea and vomiting; neurological disturbances (1.7%), primarily seizures, headaches, and sleep disturbances; and dermatologic problems (1.4%), primarily rashes. It is important to note, however, that many of these events occurred at doses that exceed the recommended daily dose.

The table below is presented solely to indicate the relative frequency of adverse events reported in representative controlled clinical studies conducted to evaluate the safety and efficacy of Wellbutrin under relatively similar conditions of daily dosage (300-600 mg), setting, and duration (3-4 weeks). The figures cited cannot be used to predict precisely the incidence of untoward events in the course of usual medical practice where patient characteristics and other factors must differ from those which prevailed in the clinical trials. These incidence figures also cannot be compared with those obtained from other clinical studies involving related drug products as each group of drug trials is conducted under a different set of conditions. Finally, it is important to emphasize that the tabulation does not reflect the relative severity and/or clinical importance of the events. A better perspective on the serious adverse events associated with the use of Wellbutrin is provided in the WARNINGS and PRECAUTIONS sections.

TREATMENT EMERGENT ADVERSE EXPERIENCE INCIDENCE IN PLACEBO-CONTROLLED CLINICAL TRIALS* (Percent of Patients Reporting)

Adverse Experience	Wellbutrin Patients (n = 323)	Placebo Patients (n = 185)	Adverse Experience	Wellbutrin Patients (n = 323)	Placebo Patients (n = 185)
CARDIOVASCULAR			Dry Mouth	27.6	18.4
Cardiac Arrhythmias	5.3	4.3	Excessive Sweating	22.3	14.6
Dizziness	22.3	16.2	Headache/Migraine	25.7	22.2
Hypertension	4.3	1.6	Impaired Sleep Quality	4.0	1.6
Hypotension	2.5	2.2	Increased Salivary Flow	3.4	3.8
Palpitations	3.7	2.2	Insomnia	18.6	15.7
Syncope	1.2	0.5	Muscle Spasms	1.9	3.2
Tachycardia	10.8	8.6	Pseudoparkinsonism	1.5	1.6
DERMATOLOGIC			Sedation	19.8	19.5
Pruritus	2.2	0.0	Sensory Disturbance	4.0	3.2
Rash	8.0	6.5	Tremor	21.1	7.6
GASTROINTESTINAL			NEUROPSYCHIATRIC		
Anorexia	18.3	18.4	Agitation	31.9	22.2
Appetite Increase	3.7	2.2	Anxiety	3.1	1.1
Constipation	26.0	17.3	Confusion	8.4	4.9
Diarrhea	6.8	8.6	Decreased Libido	3.1	1.6
Dyspepsia	3.1	2.2	Delusions	1.2	1.1
Nausea/Vomiting	22.9	18.9	Disturbed Concentration	3.1	3.8
Weight Gain	13.6	22.7	Euphoria	1.2	0.5
Weight Loss	23.2	23.2	Hostility	5.6	3.8
GENITOURINARY			NONSPECIFIC		
Impotence	3.4	3.1	Fatigue	5.0	8.6
Menstrual Complaints	4.7	1.1	Fever/Chills	1.2	0.5
Urinary Frequency	2.5	2.2	RESPIRATORY		
Urinary Retention	1.9	2.2	Upper Respiratory Complaints	5.0	11.4
MUSCULOSKELETAL			SPECIAL SENSES		
Arthritis	3.1	2.7	Auditory Disturbance	5.3	3.2
NEUROLOGICAL			Blurred Vision	14.6	10.3
Akathisia	1.5	1.1	Gustatory Disturbance	3.1	1.1
Akinesia/Bradykinesia	8.0	8.6			
Cutaneous Temperature Disturbance	1.9	1.6			

*Events reported by at least 1% of Wellbutrin patients are included.

Other Events Observed During the Development of Wellbutrin: The conditions and duration of exposure to Wellbutrin varied greatly and a substantial proportion of the experience was gained in open uncontrolled clinical settings. During this experience, numerous adverse events were reported; however, without appropriate controls, it is impossible to determine with certainty which events were or were not caused by Wellbutrin. The following enumeration is organized by organ system and describes events in terms of the relative frequency of reporting in the data base. Events of major clinical importance are also described in the WARNINGS and PRECAUTIONS sections of the labeling.

The following definitions of frequency are used: Frequent adverse events are defined as those occurring at least 1/100 patients. Infrequent adverse events are those occurring in 1/100 to 1/1000 patients, while rare events are those occurring in less than 1/1000 patients.

Cardiovascular: Frequent was edema; infrequent were chest pain, EKG abnormalities (premature beat and nonspecific ST-T changes), and shortness of breath/dyspnea; rare were flushing, pallor, phlebitis and myocardial infarction.

Dermatologic: Frequent were nonspecific rashes; infrequent were alopecia and dry skin; rare were change in hair color, hirsutism and acne.

Endocrine: Infrequent was gynecostasia; rare were glycosuria and hormone level change.

Gastrointestinal: Infrequent were dysphagia, thirst disturbance, and liver damage/ jaundice; rare were rect complaints, colitis, G.I. bleeding, intestinal perforation and stomach ulcer.

Genitourinary: Frequent was nocturia; infrequent were vaginal irritation, testicular swelling, urinary tract infection, painful erection, and retarded ejaculation; rare were dysuria, enuresis, urinary incontinence, menopausal ovarian disorder, pelvic infection, cystitis, dyspareunia, and painful ejaculation.

Hematologic/Oncologic: Rare were lymphadenopathy, anemia and pancytopenia.

Musculoskeletal: Rare was musculoskeletal chest pain.

Neurological: (see WARNINGS) Frequent were ataxia/incoordination, seizure, myoclonus, dyskinesia, or dystonia; infrequent were mydriasis, vertigo, and dysarthria; rare were EEG abnormality, abnormal neurological exam, impaired attention, sciatica and aphasia.

Neuropsychiatric: (see PRECAUTIONS) Frequent were mania/hypomania, increased libido, hallucination decrease in sexual function, and depression; infrequent were memory impairment, depersonalization, psychotic dysphoria, mood instability, paranoia, formal thought disorder, and frigidity; rare was suicidal ideation.
Oral Complaints: Frequent was stomatitis; infrequent were toothache, bruxism, gum irritation, and oral edema rare was glossitis.

Respiratory: Infrequent were bronchitis and shortness of breath/dyspnea; rare were epistaxis, rate or rhythm disorder, pneumonia and pulmonary embolism.

Special Senses: Infrequent was visual disturbance; rare was diplopia.

Nonspecific: Frequent were flu-like symptoms; infrequent was nonspecific pain; rare were body odor, surgical related pain, infection, medication reaction and overdose.

Postintroduction Reports: Voluntary reports of adverse events temporally associated with Wellbutrin have been received since market introduction and which may have no causal relationship with the drug included the following:

Cardiovascular: orthostatic hypotension, third degree heartblock

Gastrointestinal: esophagitis, hepatitis

Hemic and Lymphatic: ecchymosis, leukocytosis, leukopenia

Musculoskeletal: arthralgia, myalgia, muscle rigidity/fever/rhabdomyolysis

Nervous: coma, delirium, dream abnormalities, paresthesia, unmasking of tardive dyskinesia

Skin and Appendages: angioedema, exfoliative dermatitis, urticaria

Special Senses: tinnitus

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For free listing of your organization's official annual or regional meeting, please send us the following information: sponsor, location, inclusive dates, type and number of continuing education credit (if available), and the name, address, and telephone number of the person or group to contact for more information. All notices and changes must be received no later than 120 days before the first day of the month of desired publication and should be addressed to Calendar, American Journal of Psychiatry, 1400 K Street, NW, Washington, DC 20005; 202-682-6021 (tel), 202-682-6016 (fax). Because of space limitations, only listings of meetings of the greatest interest to Journal readers will be included.

APRIL

April 1–2, 16th Annual Symposium on Mental Health and the Law, Richmond, Va. Contact: Bettie Amiss, Administrator, Institute of Law, Psychiatry and Public Policy, Box 100, Blue Ridge Hospital, Charlottesville, VA 22901; 804-924-5435.

April 1–2, Eighth Annual Pennsylvania Conference on Schizophrenia, "Hospital to Community: Building Everyday Lives," Philadelphia. Contact: Stephen L. Schwartz, M.D., Conference Coordinator; 215-955-6503.

April 1–2, Fifth Annual Bridgewater State Hospital Conference, "The Study of Violence: A Clinical and Forensic Perspective on Violence in the Family" (12 hours of CME category I credit available), Bridgewater, Mass. Contact: Laurie Pittsley, Bridgewater State Hospital, 20 Administration Rd., Bridgewater, MA 02324; 617-727-6086, ext 388.

April 1–3, West Coast Neuropsychology Conference, "Neuropsychology With Children: Assessment and Management," University of California, San Diego. Contact: Cass Jones, Professional Conference Management, 7916 Convoy Court, San Diego, CA 92111; 619-565-9921 (tel), 619-565-9954 (fax).

April 6–10, international symposium, "Folk Medicine of Ireland" (21 hours of CME category I and 3 hours of category II credit available), sponsored by the Southern California Neuropsychiatric Institute, Dublin. Contact: Ann McCormick, SCNPI, 6794 La Jolla Blvd., La Jolla, CA 92037; 619-454-2102 (tel), 619-454-2104 (fax).

April 16, Fourth Annual Day in Psychoanalysis, "Curative Factors in Psychoanalysis—Three Perspectives on How Psychoanalytic Treatment Works," The Toronto Psychoanalytic Society, Toronto. Contact: OISE Conference Office, Suite 4-420, 252 Bloor Street W, M5S 1V6 Toronto, Ont., Canada; 416-926-4711.

April 18, Fifth Annual Symposium, "Treatment of Headaches and Facial Pain," New York Headache Center, New York. Contact: Alexander Mauskop, M.D., Director, New York Headache Center, 301 East 66th Street, New York, NY 10021; 212-794-3550.

April 22–25, 11th Annual Symposium in Forensic Psychiatry, American College of Forensic Psychiatry, Santa Fe, N.Mex. Contact: Ed Miller, Executive Director, 26701 Quail Creek, Suite 295, Laguna Hills, CA 92656; 714-831-0236.

April 23, Fourth Annual Pittsburgh Neuropsychiatry Conference, "Neural Pathways and Behavior," University of Pittsburgh Western Psychiatric Institute and Clinic, Pittsburgh. Contact: Mary McAuley, Western Psychiatric Institute and Clinic, 3811 O'Hara Street, Pittsburgh, PA 15123; 412-624-8265.

April 23–25, Ninth Annual Conference, "Demonstrating and Sharing Integrative Therapies," Society for the Exploration of Psychotherapy Integration, New York. Contact: Saul D. Raw, C.S.W., 133 Lincoln Place, Brooklyn, NY 11217-3605; 718-638-9526.

April 24–25, conference, "The Cutting Edge 1993: Treatment of Severe Personality Disorders," University of California, San Diego. Contact: Cass Jones, Professional Conference Management, 7916 Convoy Court, San Diego, CA 92111; 619-565-9921 (tel), 619-565-9954 (fax).

April 29–May 1, International Congress on the Dually Diagnosed (Mental Retardation/Mental Illness), Harvard Medical School and National Association for the Dually Diagnosed, Boston. Contact: NADD, 110 Prince Street, Kingston, NY 12401; 914-331-4336 or 800-331-5362 (tel), 914-331-4336 (fax).

April 30–May 1, conference, "Women," Cambridge Hospital, Harvard Medical School, Wellesley College, Boston. Contact: Judy Reiner Platt, Ed.D., Cambridge Hospital, 1493 Cambridge Street, Cambridge, MA 02139; 617-864-6165.

MAY

May 5–9, 11th Scientific Meeting, "Chaos and/or Community," A.K. Rice Institute, Los Angeles. Contact: Nancy Angelo, A.K. Rice Institute, P.O. Box 1776, Jupiter, FL 33468-1776; 407-744-1350 (tel), 407-744-5998 (fax).

May 19–23, meeting, American Back Society, Buffalo. Contact: Aubrey A. Swartz, M.D., Director, 2647 East 14th Street, Suite 401, Oakland, CA 94601; 510-536-9929 (tel), 510-536-1812 (fax).

May 22–27, 146th Annual Meeting, American Psychiatric Association, San Francisco. Contact: George Campbell, Director, Meetings Management, APA, 1400 K Street, NW, Washington, DC 20005; 202-682-6193.

(Continued on following page)

May 24, satellite meeting (held in conjunction with the APA annual meeting), American Society of Clinical Psychopharmacology, San Francisco. Contact: Peter Ross, P.O. Box 2257, New York, NY 10116-2257; 212-268-4260 (tel), 212-268-4434 (fax).

May 29-30, workshop, "Authenticity in Family Therapy," the Satir Professional Development Institute of Manitoba, Inc., Winnipeg, Man. Contact: Renee Frenette, 1141 Dudley Ave., R3M 1R6 Winnipeg, Man., Canada; 204-475-0303.

May 30-June 2, 16th Annual Meeting of the Canadian College of Neuropsychopharmacology, CCNP and the British Association of Psychopharmacology, Montreal. Contact: Dr. S.N. Young, Dept. of Psychiatry, McGill University, 1033 Pine Ave., W, H3A 1A1 Montreal, Que., Canada; 514-398-7317 (tel), 514-398-4370 (fax).

JUNE

June 9-11, international conference, "Chronic Diseases and Changing Care Patterns in an Aging Society," Netherlands Society for Public Health and Science, University of Amsterdam, Amsterdam. Contact: Dr. Trudi van den Bos or Wien Limburg, Institute of Social Medicine, University of Amsterdam, Meibergdreef 15, 1105 AZ Amsterdam, The Netherlands; 31-20-5664707 (tel), 31-20-6912401 (fax).

June 17-19, international conference, joint meeting of the Italian Groups for the Study of Movement Disorders and the Movement Disorder Society, Spoleto, Italy. Contact: Dr. Alfredo Berardelli, Dipartimento Scienze Neurologiche, Viale

Universita' 30, 00185 Rome, Italy; 06-49914700 (tel), 06-49914302 (fax).

June 18-19, conference, "Child Psychotherapy," Cambridge Hospital, Harvard Medical School, Boston. Contact: Judy Reiner Platt, Ed.D., Cambridge Hospital, 1493 Cambridge Street, Cambridge, MA 02139; 617-864-6165.

JULY

July 12-30, summer conference series on Women's Mental Health Issues, Malpractice Prevention, Treatment Prevention, Treatment Resistant Depression, Effective Psychiatric Interventions Using Biofeedback, the Marriage Cycle: Marriage, Divorce, and Remarriage, and Clinical Psychopharmacology. The Menninger Clinic, Steamboat Springs, Colo. Contact: Division of Continuing Education; 800-288-7377.

July 24-29, institute, "Assessment and Therapy With Suicidal Adults," "Assessment and Therapy With Suicidal Adolescents," "Self-Destructive Behavior in Borderline Patients," American Association of Suicidology, Sante Fe, N.Mex. Contact: American Association of Suicidology, 2459 S Ash, Denver, CO 80222; 303-692-0985.

AUGUST

August 22-27, Seventh World Congress on Pain, International Association for the Study of Pain, Paris. Contact: IASP, 909 NE 43rd Street, Suite 306, Seattle, WA 98105; 206-547-6409 (tel), 206-547-1703 (fax).

Books Received

- L'Enfant dans sa Famille: Le Développement en Péril**, by E.J. Anthony and C. Chiland. Paris, Presses Universitaires de France, 1992, 716 pp., no price listed.
- Adult Cardiac Surgery**, by Robert M. Bojar, M.D. Boston, Blackwell Scientific Publishers, 1992, 533 pp., no price listed.
- The Sleep Management Plan**, by Dale Hanson Bourke. New York, Harper & Row, 1990, 146 pp., \$5.50 (paper).
- The First Session in Brief Therapy**, edited by Simon H. Budman, Michael F. Hoyt, and Steven Friedman. New York, Guilford Press, 1992, 370 pp., \$35.00.
- Do What He Says! He's Crazy!!**, by John Callahan. New York, William Morrow, 1992, 125 pp., \$8.00 (paper).
- Mental Health Consultation and Collaboration**, by Gerald Caplan and Ruth B. Caplan. San Francisco, Jossey-Bass, 1993, 386 pp., \$32.95.
- Issues and Ethics in the Helping Professions**, 4th ed., by Gerald Corey, Marianne Corey, and Patrick Callanan. Pacific Grove, Calif., Brooks/Cole, 1992, 430 pp., \$29.75.
- Minna's Story: The Secret Love of Dr. Sigmund Freud**, by Kathleen Daniels. Santa Fe, N.Mex., Health Press, 1992, 177 pp., \$18.95.
- We Are All the Target: A Handbook of Terrorism Avoidance and Hostage Survival**, by Douglas S. Derrer. Annapolis, Md., Naval Institute Press, 1992, 127 pp., \$14.95 (paper).
- Clinical Interaction and the Analysis of Meaning: A New Psychoanalytic Theory**, by Theo L. Dorpat and Michael L. Miller. Hillsdale, N.J., Analytic Press, 1992, 298 pp., \$43.95.
- Crafts in Therapy and Rehabilitation**, by Margaret Drake. Thorofare, N.J., Slack, 1992, 206 pp., no price listed.
- Ego and Archetype**, by Edward F. Edinger. Boston, Shambhala, 1991, 295 pp., \$15.00 (tape).
- Nature's Mind: The Biological Roots of Thinking, Emotions, Sexuality, Language, and Intelligence**, by Michael S. Gazzaniga. New York, Basic Books, 1992, 214 pp., \$25.00.
- Beyond Transference: When the Therapist's Real Life Intrudes**, edited by Judith H. Gold and John C. Nemiah. Washington, D.C., American Psychiatric Press, 1992, 175 pp., \$26.00.
- The Wounded Healers: Creative Illness in the Pioneers of Depth Psychology**, by Marvin Goldwert. Lanham, Md., University Press of America, 1992, 164 pp., \$39.75; \$19.50 (paper).
- Finishing Well: Aging and Reparation in the Intergenerational Family**, by Terry D. Hargrave and William T. Anderson. New York, Brunner/Mazel, 1992, 222 pp., \$26.95.
- Berthe Morisot's Images of Women**, by Anne Higonnet. Cambridge, Mass., Harvard University Press, 1992, 299 pp., \$45.00.
- Intensive Treatment of the Homeless Mentally Ill**, edited by Steven E. Katz, David Nardacci, and Albert Sabatini. Washington, D.C., American Psychiatric Press, 1992, 207 pp., \$18.50 (paper).
- Who Plays? Who Pays? Who Cares?**, by Sylvia Kenig; Ray Elling, series editor. Amityville, N.Y., Baywood, 1992, 213 pp., \$21.75 (paper).
- From Anger to Forgiveness**, by Earnie Larsen. New York, Ballantine Books, 1992, 194 pp., \$4.99 (paper).
- Fundamentals of Psychopharmacology**, by B.E. Leonard. New York, John Wiley & Sons, 1992, 253 pp., \$32.95.
- In Defense of Schreber: Soul Murder and Psychiatry**, by Zvi Lothane. Hillsdale, N.J., Analytic Press, 1992, 523 pp., no price listed.
- International Review of Health Psychology**, vol. I, by S. Maes, H. Leventhal, and M. Johnston. New York, John Wiley & Sons, 1992, 224 pp., no price listed.
- The Windows of Experience: Moving Beyond Recovery to Wholeness**, by Thomas Patrick Malone and Patrick Thomas Malone. New York, Simon & Schuster, 1992, 408 pp., \$23.00.
- Testing and Your Child: What You Should Know About 150 of the Most Common Medical, Educational, and Psychological Tests**, by Virginia McCullough. New York, Plume, 1992, 316 pp., \$12.00 (paper).
- Practical Clinical Hypnosis: Techniques and Applications**, by Robert G. Meyer. New York, Lexington Books, 1992, 404 pp., \$35.00.
- Understanding and Preventing Violence**, edited by Albert J. Reiss, Jr., and Jeffrey A. Roth. Washington, D.C., National Academy Press, 1993, 438 pp., \$49.95.
- The Male Paradox**, by John Munder Ross. New York, Simon & Schuster, 1992, 339 pp., \$23.00.
- Alcoholism and the Family**, edited by Satoru Saitoh, Peter Steinglass, and Marc Schuckit. New York, Brunner/Mazel, 1992, 322 pp., \$25.00 (paper).
- Cognitive Science and Clinical Disorders**, edited by Dan J. Stein and Jeffrey E. Young. New York, Academic Press, 1992, 388 pp., no price listed.
- The Ethics of Authenticity**, by Charles Taylor. Cambridge, Mass., Harvard University Press, 1992, 135 pp., \$17.95.
- Handbook of Pain Assessment**, edited by Dennis C. Turk and Ronald Melzack. New York, Guilford Press, 1992, 491 pp., \$60.00.
- Working With Culture: Psychotherapeutic Interventions With Ethnic Minority Children and Adolescents**, edited by Luis A. Vargas and Joan D. Koss-Chiomo. San Francisco, Jossey-Bass, 1992, 309 pp., \$29.95.
- The History of Clinical Psychology in Autobiography**, vol II, edited by C. Eugene Walker. Pacific Grove, Calif., Brooks/Cole, 1992, 284 pp., \$50.00.
- The Chemically Dependent: Phases of Treatment and Recovery**, by Barbara Wallace. New York, Brunner/Mazel, 1992, 384 pp., \$45.00.
- Ordinary Magic: Everyday Life as a Spiritual Path**, edited by John Welwood. Boston, Shambhala, 1992, 338 pp., \$13.00 (paper).
- Schizophrenic Disorders: Sense and Nonsense in Conceptualization, Assessment, and Treatment**, by Leighton C. Whitaker. New York, Plenum, 1992, 247 pp., \$35.00.
- Principles and Practice of Relapse Prevention**, edited by Peter H. Wilson. New York, Guilford Press, 1992, 383 pp., \$35.00.
- Leaving My Father's House: A Journey to Conscious Femininity**, by Marion Woodman. Boston, Shambhala, 1992, 352 pp., \$16.00 (paper).
- Hypnosis and the Treatment of Depressions: Strategies for Change**, by Michael D. Yapko. New York, Brunner/Mazel, 1992, 222 pp., \$26.95.
- Treating the Borderline Patient: A Contract-Based Approach**, by Frank E. Yeomans, Michael A. Selzer, and John F. Clarkin. New York, Basic Books, 1992, 204 pp., \$32.00.

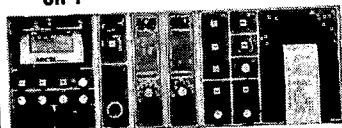
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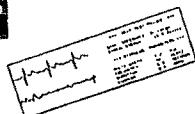
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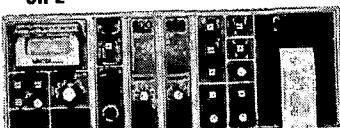
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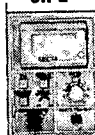
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Editorial

Practice Guidelines

This issue of the *Journal* contains the first practice guideline to be approved by APA. The term "practice guideline" refers to a set of patient care strategies developed to assist physicians in clinical decision making. Several more guidelines are being prepared and will be submitted for publication in the *Journal* as they are completed. Although APA has published specific recommendations about the practice of psychiatry since 1851, the current commitment of resources to the practice guideline process represents a qualitative change in APA's role in establishing guidelines. Such a change raises many questions.

Why Is APA Developing Practice Guidelines?

For nearly 150 years, APA's fundamental aim in developing practice guidelines has been to assist psychiatrists in their clinical decision making, with the ultimate goal of improving the care of patients. The explosion of knowledge in our field over the last several decades amplifies the value of these guidelines. Furthermore, the current health care climate is characterized by rising concerns about quality of care, access to care, and cost. Efforts to respond to these problems by exerting external control over the types and amount of care that can be provided have led to new concerns about the quality of the data on which such efforts are based and the process by which the data are used to determine "appropriate" or "reimbursable" care. The realization that both treatment and reimbursement decisions are occurring, without systematic scientific and clinical input, has led APA, along with many other medical specialty societies, to accelerate the process of documenting clearly and concisely what is known and what is not known about the treatment of patients. While there are a number of other entities, including the federal government (through the Agency for Health Care Policy and Research), that are also writing practice guidelines, APA has decided that the psychiatric profession should take the lead in describing the best treatments and the range of appropriate treatments available to patients with mental illness.

How Is APA Going About This Task?

APA has established the Steering Committee on Practice Guidelines, chaired by John S. McIntyre, M.D. The steering committee decides on topics for guidelines by using the following criteria: 1) degree of public importance, 2) relevance to psychiatric practice, 3) availability of information and relevant data, 4) availability of work already done that would be useful in the development of a practice guideline, and 5) degree to which increased psychiatric attention to and involvement in the area would be helpful for the field. Once a topic is chosen, a work group is formed to draft the guideline. By design, the work group consists of psychiatrists in active clinical practice with diverse expertise and practice experience relevant to the topic. Policies established by the steering committee guide the work of systematically reviewing data in the literature and forging consensus on the implications of that data, as well as describing a clinical consensus. These policies, in turn, stem from criteria formulated

by the American Medical Association to promote the development of guidelines that make optimal use of evidence derived from the literature and of clinical consensus. The guideline is written in successive drafts, each being revised on the basis of comments received from an increasing number of people. Early drafts are sent to the Steering Committee and about 50 expert reviewers; later drafts are sent to members of the APA Assembly, the district branches, the Board of Trustees, and other APA components. Drafts are available to any APA member by request through their district branch. In addition, individual experts who are not APA members, along with relevant professional, scientific, and patient organizations, are asked to review the drafts. Once all comments have been considered, a final draft is sent to the Assembly and Board of Trustees for their approval. Thus the Practice Guideline for Eating Disorders and successive guidelines will be reviewed by hundreds of psychiatrists and other interested parties before their finalization.

What Are the Anticipated Benefits of This Project?

Ultimately, the aim of practice guidelines is to improve patient care. Although some have argued that no guidelines should be promulgated until "all the data are in," this is not possible given the pressure of clinical and administrative decisions. Psychiatrists and those charged with the allocation of health care resources must try to make the best possible decisions on the basis of currently available data. Guidelines should help practicing psychiatrists determine what is known today about how best to help their patients.

Toward the end of helping patients, guidelines are expected to have other beneficial effects. They are a vehicle for educating psychiatrists, other medical and mental health professionals, and the general public about appropriate and inappropriate treatments. By demonstrating that the quality of evidence for psychiatric treatments is on par with (and in many cases exceeds) that for other medical care, they will contribute to the credibility of the field. Guidelines will identify those areas in which critical information is lacking and in which research could be expected to improve clinical decisions. Finally, guidelines might help those charged with overseeing the utilization and reimbursement of psychiatric services to develop more scientifically based and clinically sensitive criteria.

What Are the Anticipated Risks of This Project?

APA anticipates that the risks of this project are small and are considerably outweighed by the benefits. One risk is that the guidelines will be misinterpreted and misused by third parties in a way that will ultimately harm patients. Although this risk rightfully concerns many psychiatrists, it is the judgment of the steering committee, the Assembly, and the Board of Trustees that the existence of guidelines will help clarify the sources of disagreement between treating psychiatrists and reviewers and will be a great improvement over the use of "secret" criteria or criteria developed through less objective procedures. In addition, many have expressed the concern that guidelines will "homogenize" the care of patients and will detract from psychiatrists' freedom to shape treatment in ways that they feel best suit their individual patients. Inevitably, there is a tension in writing guidelines between the desire for specificity and the desire to allow for the consideration of individual clinical circumstances. These concerns must be balanced in such a way that allows psychiatrists to make appropriate clinical decisions. Finally, there are concerns that APA-approved guidelines will lead to an increase in malpractice claims. In fact, those medical specialties which have been developing guidelines over the past decade report that guidelines seem to have had the positive effect of fewer claims and, for at least one specialty, lower premiums.

How Can We Improve the Current Process?

Practical considerations. At present, the initial draft of a guideline is written by a work group, with support from the APA Office of Research. The interest and participation of many APA members have been gratifying and are absolutely essential

to the development of a quality document. The amount of time and resources required to complete a guideline, however, is large, and ways to increase the efficiency of the process must be explored.

Efforts to gather clinically useful data. Guidelines should be based on objective data whenever possible. Systematic reviews of the literature are an essential part of this work. However, efforts to synthesize studies in any given area are hampered by the uneven quantity and quality of studies; by problems in generalizing from a literature largely derived from tertiary care research settings to more typical clinical practice; by the inherent difficulty in conducting controlled studies of some treatments for some populations; and by the difficulty in characterizing "clinical consensus." These issues were recently addressed in a conference presented by APA, "Challenges in Developing Psychiatric Practice Guidelines." One recommendation of the conference was that APA form a practice research network. Such a network would involve a panel of perhaps 1,000 psychiatrists in office-based settings who would cooperate to gather data and conduct clinical research. Practice research networks have been used in other areas of medicine (e.g., family practice and pediatrics) to gather data from practice settings of relevance to the development of guidelines. For example, data about prevailing practice patterns and patient outcomes could be systematically gathered and incorporated into the guidelines. In addition, the impact of guidelines on psychiatric practice and patient outcomes could be assessed (perhaps testing different education or dissemination strategies); ultimately, it should be possible to determine whether guidelines improve patient care.

Efforts to maintain up-to-date guidelines. The guidelines are, of course, only as good as the quality and currency of the data on which they are based. The current plan is to update guidelines every 3 to 5 years. As the number of guidelines grows, the need for a system to track new developments and efficiently revise guidelines will be increasingly important. APA is exploring various technologies for dissemination of guidelines that may facilitate these efforts.

Conclusions

The Practice Guideline for Eating Disorders represents the first step in a process that we believe will be of enormous importance for American psychiatry as we move into the second 150 years of APA. We also hope that this will be an iterative process actively involving a large proportion of our membership. We sincerely welcome suggestions and comments.

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Cognitive Remediation in Schizophrenia: Is It Time Yet?

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Considerable effort has been directed toward identifying and conceptualizing information-processing deficits in schizophrenia and other psychotic disorders. The impressive gains in this field have made meaningful contributions to our understanding of the etiology and course of these disorders. This article considers whether the time is ripe to move beyond identification of these deficits to remediation of them. This move is far from a simple matter; it requires careful attention to theoretical frameworks and to the criteria for selecting certain cognitive deficits, among many, for remediation. A sparse and somewhat dated literature suggests that certain types of cognitive remediation, at least when defined in a narrow sense, are feasible with schizophrenic patients. This literature can reasonably justify continuing investigations on a modest scale. Specific questions for further studies include the following. 1) Which cognitive models provide a framework to guide cognitive interventions? 2) Which specific functions or levels of information processing should be targeted by cognitive interventions? 3) Are cognitive interventions effective? 4) Does the remediation of basic cognitive deficits generalize? A major strength of this area of investigation is the testability of the questions. Several research designs are tentatively suggested.

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For the most part, research on information processing in schizophrenia has been devoted to conceptualizing and identifying key cognitive deficits in an effort to understand better the etiology and course of the disorder. This commentary considers whether the time is ripe to move beyond *identification* of cognitive deficits to *remediation* of them. I hope that a broad and somewhat speculative discussion of cognitive remediation issues will serve a heuristic function. Although the vast majority of data in this area have been collected from patients with a schizophrenic diagnosis, cognitive remediation is probably egalitarian in its effects. Hence, much of the discussion is likely to apply to psychotic disorders in general.

As investigators try to isolate "nuclear" or "central"

cognitive deficits in schizophrenia, several conceptual and methodological problems arise. At a practical level, it is all too easy to uncover cognitive deficits. Schizophrenic patients, as a group, perform poorly on nearly every cognitive measure. Thus, merely showing that patients differ from normal control subjects on a certain task is not very informative. On a given task, only a subgroup (often about half) of schizophrenic patients will perform in the deficit range (1). However, it is uncommon for chronic patients not to show any deficits across a battery of neuropsychological tests.

Despite the rather large number of documented deficits in schizophrenia, a characteristic cognitive profile has yet to emerge. This impressive variability occurs partly because the cognitive deficits can be of two general types: those that are primary and central to the disorder and those that are secondary and result from the psychotic episode (perhaps because of intrusive symptoms or side effects of pharmacological treatments). This distinction between primary and secondary deficits is central to a vulnerability/stress model, and several paradigms have been developed to separate pri-

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mary from secondary deficits (sometimes called epiphenomena).

For example, patients can be tested in longitudinal designs across differing clinical states to distinguish between cognitive deficits that are episode indicators (i.e., are present only during psychotic episodes) and those that are vulnerability indicators (i.e., are stable across clinical states) (2–4). Deficits that are present in remission are presumed to reflect a relatively enduring vulnerability to the disorder. In addition, a subgroup of the patient's first-degree relatives are expected to be vulnerable to the disorder (i.e., to have the genetic predisposition and its associated cognitive manifestation), even if they do not show psychotic symptoms, perhaps because of protective environmental and biological factors. Hence, a cognitive deficit that is found during periods of remission and found disproportionately in first-degree relatives is likely to be a vulnerability indicator.

Another paradigm that has been used to distinguish between primary and secondary deficits is the high-risk design. High-risk projects assess subjects who are more likely than the general population to develop schizophrenia, such as the offspring of schizophrenic mothers (5, 6). The investigation begins before subjects enter their age of risk for schizophrenia to identify cognitive, behavioral, or psychophysiological abnormalities prior to the initial episode. In some studies, the high-risk group members are tested repeatedly as they pass through their age of greatest risk for the disorder. Eventually, a subgroup of the high-risk children will develop schizophrenia or other behavioral disorders, and investigators can refer back to the previous testings to identify cognitive or psychophysiological patterns that distinguish these children from those who do not develop psychopathology (7).

The high-risk design is particularly valuable because any deficits that are uncovered occur before the onset of the disorder and cannot easily be explained as a consequence of the symptoms. The work of Erlenmeyer-Kimling, Cornblatt, and colleagues (6–8) provides hypotheses about ways in which an early cognitive deficit can contribute to the onset of the disorder. They found that when children of schizophrenic mothers were originally tested (mean age=9.1 years), they had poorer performance on the Continuous Performance Test, which is a measure of vigilance and signal-to-noise discrimination (6). It is interesting that the high-risk children who later developed psychiatric problems performed worse on the Continuous Performance Test than the remainder of the high-risk group. The attention deficits assessed by the Continuous Performance Test might create an experience of sensory overload, which is sometimes reported before a psychotic episode. This experience could, in turn, lead to social and emotional isolation and a resulting deficit in coping skills (8). It seems possible, at least in theory, that high-risk studies could not only identify individuals at greatest risk but also offer an empirical basis for directing preventive interventions that might reduce the likelihood of having an initial episode.

Substantial efforts have gone into studying the nature and characteristics of information-processing deficits in schizophrenia. Moreover, considerable progress has been made in specifying information-processing deficits that seem to reflect enduring vulnerability to psychotic disorders. Despite these gains in identifying information-processing deficits, remarkably little effort has been devoted to their remediation. To illustrate this point, an informal survey was conducted of four prominent psychopathology journals (*American Journal of Psychiatry*, *Archives of General Psychiatry*, *Journal of Abnormal Psychology*, and *Schizophrenia Bulletin*) for a 3-year period (1987–1989). During the survey period, these journals published a total of 45 articles that addressed issues of information processing in psychotic disorders. Of these 45 articles, only one addressed the issue of cognitive remediation (in the sense that in the study an attempt was made to modify the performance of the patients). The survey was not intended to be a rigorous assessment of the field but to demonstrate that the issue of remediation is largely neglected. The neglect of remediation is puzzling when one considers the pervasiveness of cognitive deficits in schizophrenia and the role that the deficits seem to play in contributing to the onset of psychotic episodes and restricting the functioning of patients, even in periods of remission. Given these considerations, such an omission is, at the very least, regrettable.

LESSONS FROM NEUROPSYCHOLOGY

Cognitive remediation (the restoration of cognitive functioning) can be considered an area of intersection among the disciplines of clinical neuropsychology, rehabilitation, and behavioral psychology. Although cognitive remediation has been extensively explored with brain-injured patients, the impact for psychotic patients has been minimal. Such a failure to cross discipline boundaries is perhaps understandable because the broader field of clinical neuropsychology has had remarkably little impact on psychiatric treatments. For much of the past century, the efforts of clinical neuropsychologists have been devoted to relatively unsuccessful attempts to discriminate between psychotic and brain-damaged patients on the basis of neuropsychological tests (9, 10). Considering the current emphasis on understanding schizophrenia in terms of a brain-injury or neurodevelopmental model, the lack of success in discriminating groups of patients is hardly surprising. Despite this largely unproductive history, neuropsychological assessment potentially offers the basis for a well-grounded remediation program for psychiatric patients (11). In addition, neuropsychology has developed several models for rehabilitation that might be applicable to psychiatric patients. After years of trying to separate psychotic from brain-damaged patients, it would be ironic if clinical neuropsychology's major contribution to psychiatric treatment were to demonstrate commonality between these populations in terms of their response to cognitive remediation.

The techniques of cognitive remediation with brain-injured individuals vary widely, and several treatment models have been proposed (12, 13). To simplify these models, this discussion focuses on three basic approaches: general stimulation, substitution-transfer, and behavior modification. Other approaches have been used successfully for rehabilitation of global functions such as social skills (14, 15), but this discussion emphasizes techniques used in the remediation of relatively elementary cognitive functions.

The general stimulation approach requires that the patient perform repetitive drills and exercises in an attempt to remedy a deficiency. Often the tasks that were used to identify deficits (e.g., from a neuropsychological evaluation) also serve as the exercises for remediation of the deficits with general stimulation. When the dependent measure serves this dual role, it provides excellent monitoring of the patient's progress, but it also runs the risk of confusing the dependent measure with the underlying construct that the measure is supposed to reflect. Although this approach is practical and probably the most commonly used (16), empirical support for generalization of the results outside of the clinic is not impressive (17). In fact, there is some evidence that practice alone does not produce improvement. For example, Wilson (18) gave 6 weeks of intensive memory training with the use of general stimulation to a patient with amnesia, but no improvement in general memory was noted. Similarly, Prigatano et al. (19) exposed head-injured patients to an intensive general stimulation rehabilitation program for 6 hours a day, 4 days a week, for about 6 months. They found that the patients in the training program showed an increase of about 1 item score on a test of logical memory—a paltry increase after 625 hours of training.

The substitution-transfer approach provides the patient with alternative strategies for achieving goals. Examples of this approach include the functional adaptation model (20) and the extensive work of Luria and colleagues (21). The aim of the approach is to have an intact part of the brain take over the functions of the damaged region. For example, in memory rehabilitation, patients with defective verbal memory might be trained to use external aids such as checklists or to use visual imagery as a mnemonic device. Instructing patients in alternative strategies (e.g., training in verbal mediation of problem solving) is another example of this approach.

The substitution-transfer approach has been useful under certain conditions with brain-injured patients (20), and it has been used with some success with the elderly (22, 23). For memory rehabilitation, normal elderly persons provide an excellent comparison group for schizophrenic patients because the memory deficits are not as absolute as with amnesic patients. Robertson-Tchabo et al. (22) found that normal elderly subjects could learn and apply a mnemonic device (method of loci) to increase significantly their recall of a list of high-imagery nouns. Work by Yesavage (23) has shown that substitution-transfer techniques for face and name

recall can be successful with normal elderly persons. Subjects who received the training (based on high-imagery transformations of names and prominent facial features) showed a significantly greater improvement from pre- to posttest (from 38% to 69% correct) than the control group (from 34% to 44% correct). An additional finding from this study has direct implications for training with psychotic patients: the amount of improvement was correlated with reduction in anxiety, suggesting that relaxation training might be a worthwhile adjunct to cognitive remediation.

The third approach uses behavioral learning principles (e.g., reinforcement, response cost, modeling, shaping) to improve performance. It has shown some efficacy, especially when brain injury results in severe intellectual or behavioral disturbance (16). For example, this approach has shown excellent results with mentally retarded individuals (24). A behavioral intervention also seems relevant to schizophrenia, because the approach lends itself to situations in which processing structures are intact but motivational aspects (including putative motivational structures) are lacking. Historically, there has been speculation that brain-damaged patients *cannot* perform adequately on cognitive measures because of structural limitations, and psychotic patients *will not* perform adequately because of motivational limitations (9). This speculation may have some merit, but the distinction is somewhat problematic. First, behavioral interventions have been shown to be effective with brain-damaged patients (12). Second, motivational systems are rooted in anatomical systems (25).

The role of motivation in the cognitive performance of psychotic patients attracted considerable interest in previous decades (26, 27), but in recent years it has been treated mainly as a "nuisance" variable and has rarely been subjected to systematic investigation. This neglect is unfortunate, because motivation often appears to be the most critical uncontrolled variable in the testing situation and could account for much of the individual differences in performance. The level of motivation is a critical factor to consider when one attempts to apply cognitive interventions that have been developed with brain-injured persons (who tend to be motivated) to psychotic persons (whose motivation is suspect). The paucity of recent investigations in this area might reflect the opinion that there are no "hard science" methods for studying motivation in the testing situation. Perhaps newer metabolic imaging technologies such as positron emission tomography will provide alternative ways to investigate the anatomical substrates of motivation. For example, subjects could undergo imaging while they perform the same task with and without reinforcement to determine whether specific anatomical structures will show differences in metabolic activity as a result of this motivational manipulation.

Two of the approaches I have mentioned (substitution-transfer and behavior modification) have demonstrated effectiveness with brain-injured patients under certain conditions, but are they applicable to psychotic patients?

Unfortunately, few attempts have been made to apply these techniques to psychiatric patients. Several exceptions are discussed later in this article (26–34). Suffice it to say that the studies with psychotic patients offer encouragement that cognitive deficits can be modified, at least in a limited way. Why, then, have investigators and clinicians been so reluctant to apply cognitive remediation to schizophrenia? Spring and Ravdin (35) speculated that cognitive remediation's chilly reception is due to several factors, including 1) concerns about generalization of gains in elementary processes, assessed in a laboratory, to meaningful aspects of daily functioning, 2) the belief that macro-level psychosocial interventions, such as skills training programs, will improve elementary cognitive functions, and 3) the view that the cognitive deficits are central and therefore not remediable. To these factors one can add the belief that the cognitive deficits are strictly derived from psychotic symptoms, in which case administration of neuroleptics would be sufficient for cognitive remediation. On the basis of the few data that are available, there is no reason to conclude that remediation techniques are inappropriate for psychotic patients. However, many of the fundamental questions regarding this application have not been adequately assessed.

Before we can determine whether cognitive remediation will play a meaningful role in the treatment of schizophrenia, four fundamental questions should be addressed: 1) which cognitive model (or models) provides a framework to guide cognitive interventions? 2) which specific functions or levels of information processing should be targeted by cognitive interventions? 3) are cognitive interventions effective in the remediation of the relevant deficits? and 4) does the remediation of basic cognitive deficits generalize to an improved quality of life? Each of these questions is discussed separately.

MODELS OF INFORMATION PROCESSING

Two separate but overlapping frameworks have been used to view cognitive functioning in psychotic disorders: capacity models and stage models (36). These are not the only two available models, but they have received the lion's share of the interest in psychopathology. Capacity models emphasize an individual's overall processing capacity, which is viewed as a limited resource that can be drawn upon for performing cognitive tasks (37). The amount of the resource varies both between and within individuals (e.g., because of fluctuations in levels of arousal). When cognitive deficits in psychosis are viewed from the point of view of a capacity model, they are attributed to deficiencies in the amount of an available processing resource (3). These deficiencies could be due to a reduction in central capacity (possibly owing to abnormal levels of arousal) or to inefficient allocation of the available processing resources.

In contrast to capacity models, stage models emphasize a sequential series of processing stages. The output from one stage is fed to a subsequent stage, where the information becomes transformed or elaborated.

Hence, the degree of information processing increases progressively with each step. Within a stage model, cognitive deficits in psychotic disorders are attributed to a dysfunction in an early stage of processing. In fact, many of the information-processing models in schizophrenia have postulated an "input dysfunction" that eventually leads to psychotic ideation (38). The assumption with this type of model is that a defective early stage feeds poorly processed information to the subsequent stages, and therefore later processing is disrupted because of a cascading effect (39).

The two models have considerable overlap but quite different emphases. Capacity models emphasize capacity limitations of a central processor, and they lead to a search for measures of overall processing resources and allocation strategies. In contrast, stage models emphasize the capacity limitations of a certain step or structure, and they lead to a search for dysfunctional stages of processing (40). Both models have received substantial empirical support, and the object is not to choose between them. In fact, some theorists (41) would argue that both of these models are versions of a more general limited-capacity model in which the processing limitation is placed at different structures (early versus late stages). Nonetheless, each model carries with it certain interpretations and treatment implications.

Certain experimental paradigms differentially lend themselves to interpretation within one model or another. Dual-task paradigms, in which subjects divide their attention between a primary and a secondary task, are usually interpreted within a capacity model. Alternatively, the backward masking paradigm, in which subjects identify a briefly presented target prior to visual masking, is usually viewed within a stage model.

An appropriate theoretical framework is critical for the more applied problem of remediation of cognitive deficits in psychoses. Suppose we observe a certain cognitive deficit in schizophrenic patients that appears to be a primary deficit. The choice of cognitive model will determine both our conceptualization of that deficit and the intervention. If a given stage is seen as dysfunctional, then efforts should be directed at enhancing (through general stimulation or behavioral techniques) or bypassing (through substitution-transfer techniques) that processing stage. If, on the other hand, we view the deficit as reflecting an unusually limited processing capacity, then remediation efforts should be directed at making more efficient use of the available resources, regardless of the stage of intervention (42). Reduction in anxiety, which was correlated with memory improvement in the normal elderly (23), could be interpreted within a capacity model as a means to increase processing capacity.

DIRECTING THE COGNITIVE INTERVENTION

How do we decide which cognitive deficits are most worthy of remediation efforts? The choice is difficult because schizophrenic patients, on average, show defi-

cits on nearly every information-processing measure. Two experimental approaches seem to offer empirical and logical justifications for selecting appropriate intervention targets. One approach considers "rate-limiting factors" by examining the information-processing correlates of skills training or social competence. (I return to this approach in later sections.) Another approach involves studies of vulnerability indicators and of high-risk populations.

The distinction between episode and vulnerability indicators may help direct cognitive remediation efforts, depending on which assumptions are made. Let us first consider episode indicators. If a cognitive deficit is an episode indicator and causes the psychiatric symptoms (i.e., the cognitive dysfunction temporally precedes the relapse), we could argue that cognitive intervention would directly aid symptom reduction or prevent symptom exacerbation. Episode indicators that result from psychiatric symptoms (i.e., temporally follow relapse) would not receive intervention. Unfortunately, we currently lack information on one of the most basic questions in experimental psychopathology: the temporal sequencing of episode indicators and psychotic relapse.

Alternatively, perhaps rehabilitation efforts should selectively target vulnerability indicators that exist prior to onset and in the remitted state. Most of the measures that have been proposed as vulnerability indicators have used extremely brief tachistoscopic presentations (e.g., the Continuous Performance Test, Span of Apprehension, backward masking) or have evaluated electrophysiological responses to stimuli (evoked potentials, smooth pursuit eye movements). However, cognitive rehabilitation efforts have rarely targeted these early perceptual and registration processes that seem to endure into remission.

Data from the high-risk studies raise the possibility that many of the putative vulnerability indicators not only are correlates of psychosis but might also contribute directly to the onset of the disorder (6-8). Certainly, a host of other situational and historical factors influence whether or not symptoms appear, but identification of a specific contributing factor (such as an information-processing deficit) offers direction for intervention. Perhaps remediation of these deficits could reduce the probability of having an initial episode. Hence, the findings from high-risk studies might eventually serve the dual purpose of identifying individuals at risk for psychosis as well as pinpointing the nature of the cognitive intervention that is needed.

Questions remain about whether the interventions should be directed at a broad or narrow range of cognitive abilities. Clearly, schizophrenic patients suffer from cognitive disabilities ranging widely from the elementary (such as perceptual processes) to the moderately complex (such as problem solving and verbal memory) to the highly complex (such as social perception, social schema, and communication). These different levels of cognitive abilities could be considered relatively independent subsystems or modules (43). Should we intervene at the most basic level and expect the other

functions to fall into line? The stage model would predict that the optimal intervention should be directed at the earliest dysfunctional stage, because disruption at this stage would generate later-stage problems. Alternatively, should we intervene at a more global stage such as social schema (44)? Such an intervention would be more compatible with a capacity model, in which the goal of intervention would be to maximize the available processing capacity.

Spring and Ravdin (35) asserted that cognitive intervention should range from micro-level to macro-level. The assertion seems quite reasonable but remains largely untested. In many respects, the choice of level(s) for intervention depends on the extent to which improvements in one level will generalize to another. This issue is discussed in a separate section.

ARE THE RELEVANT DEFICITS REMEDIABLE?

Once the relevant deficits or stages for intervention have been identified, the problem becomes how to reduce or eliminate the deficits. I have already discussed a range of approaches from neuropsychology that have shown some efficacy in brain-injured patients. What is the evidence to suggest that they are appropriate for psychotic patients? A series of older studies found improvement in the reaction time of schizophrenic patients when aversive shocks were used in behavior modification paradigms (26, 27, 45). These studies were designed to manipulate the level of motivation in patients. Similarly, Meiselman (46) trained chronic schizophrenic patients on single- and dual-modality reaction time tasks (using auditory and visual stimuli). Two groups of patients were studied: an experimental group who received contingent reinforcement and knowledge of results (i.e., informational feedback) and a control group who received only repeated administrations. The experimental group showed substantial improvement in utilization of dual-modality cues: the difference in reaction time between the single- and dual-modality conditions was reduced across training sessions (from 198 to 32 msec). In contrast, the difference between the single and dual conditions remained stable for the control group (from 168 to 166 msec). The results were interpreted to indicate that schizophrenic patients could broaden their cue utilization under certain motivational manipulations.

The findings from studies of event-related potentials have been mixed. A group of Japanese researchers (47) had success in increasing the amplitude of the P300 wave in a subgroup of schizophrenic patients by using a "coaching method." However, another study (48) reported that the reduced P300 amplitude in schizophrenic patients did not respond to monetary reinforcement.

In addition to the studies that followed behavioral procedures, some studies have emphasized organizational strategies. Schizophrenic patients consistently show memory deficits (49), especially on recall tasks in

comparison with recognition tasks (50, 51). The recall deficits in schizophrenia are thought to result from poor organization of information. Hence, interventions that require subjects to process information more thoroughly (e.g., to the semantic level) have produced enhanced memory performance. For example, Koh et al. (52) reported that schizophrenic patients showed a memory deficit when asked to freely recall lists of words (means were 9.81 and 13.87 words for patients and control subjects, respectively). However, after the words were sorted according to pleasantness, the schizophrenic patients did not differ significantly from the control subjects (means were 19.37 and 23.00 words, respectively, for incidental recall and 15.25 and 14.31 words, respectively, for cued recall).

At a somewhat more global level, complex problem solving has also showed encouraging plasticity in psychotic patients. The Wisconsin Card Sorting Test is a commonly used measure of concept formation and cognitive flexibility. An initial report found that schizophrenic patients improved while receiving detailed instructions, but their performance dropped to baseline levels once instructions were withdrawn. The conclusion was that schizophrenic patients were unable to learn this task (53). Three more recent studies (29, 31, 54) have had more success with improving patients' performance on this task. One of the studies (54) reported improvement with monetary reinforcement alone, but the other two (29, 31) found monetary reinforcement alone to be ineffective and relied on combinations of monetary reinforcement and detailed instructions (substitution-transfer model) to improve performance. Bellack et al. (29) found that the gains in performance were maintained on the following day. However, the stability of the training effects over longer periods of time is unknown. Recent data from our laboratory suggest that the benefit of training is smaller but still significant after 1 week (55). Also unknown is the effect of training for the Wisconsin Card Sorting Test on more important aspects of daily functioning.

Curiously, almost no study has used cognitive techniques for the remediation of a putative vulnerability indicator. One possible exception is a study by Bellissimo and Steffy (56) that attempted to modify the well-documented reaction time crossover effect but was not really aimed at improving overall performance. Another possible exception is a study by Levin et al. (57) that modified the characteristics of the target during a pursuit eye movement task. They found that all subjects' tracking improved with the manipulations, but the difference between schizophrenic patients and normal subjects remained.

Investigators may be reluctant to target a vulnerability indicator because of an erroneous notion that a putative vulnerability deficit must also be a refractory deficit. This question is strictly empirical, not deductive. Given the results from a sparse and rather dated literature, one might expect vulnerability deficits to be malleable. However, the appropriate feasibility studies are still begging to be done. Recent data from our labo-

ratory demonstrated that the performance of schizophrenic patients on the Span of Apprehension, which is a vulnerability indicator, was modifiable with the use of a combination of instructions and reinforcement. In addition, the effects were still apparent after 1 week (unpublished 1991 paper by R.S. Kern et al.).

In summary, the field of psychopathology can boast of few feasibility studies on the efficacy of cognitive remediation. Several projects conducted in the 1950s and 1960s demonstrated that reaction time was responsive to behavioral interventions. More recent studies have demonstrated improvements in complex problem solving. Despite the paucity of studies, there seems to be no basis for pessimism about the short-term responsiveness of cognitive deficits in schizophrenic patients to intervention.

DOES REMEDIATION OF COGNITIVE DEFICITS GENERALIZE?

The ultimate goal of a rehabilitation effort is to improve the functioning and quality of life of psychiatric patients. This goal seems especially relevant for cognitive intervention because cognitive deficits are thought to interfere with adaptive behavior and to hamper the acquisition of new information (58). Fundamental questions include whether improvement in elementary cognitive functioning leads to gains in adaptive functioning ("upward generalization") or whether improvements in global functions have a beneficial effect on elementary processes ("downward generalization"). Let us first consider downward generalization. Kraemer et al. (32) found that the cognitive training subprogram of Brenner et al. (30, 59) improved elementary cognitive functioning indexed by reaction time, the Continuous Performance Test, and the Span of Apprehension task. The effects were nonspecific because a social skills training program resulted in similar improvements, but the findings suggest that a downward generalization of training might occur. Olbrich and Mussgay (33) used a training procedure that involved practicing tasks of medium complexity (similar to the type of tasks used by Kraemer et al., e.g., reasoning and concept formation, verbal and visual recall). Training on these tasks improved performance on complex cognitive tasks (Embedded Figures Test, letter cancellation), but in contrast to the previous study, the improvement did not generalize to elementary cognitive tasks.

In reference to upward generalization, it seems reasonable to expect that cognitive impairment begets functional impairment, although the empirical support for this assumption is quite limited (11). In an early study, Wagner (28) used contingent reinforcement to train schizophrenic patients on attention (with a matching-to-sample task) and/or abstraction (with a type-of-categories test). Not only did the patients improve on the training tasks, but improvement with both forms of training seemed to transfer to more global intellectual measures such as similarities, proverbs, and vocabulary

tasks. A control group (well-matched for amount of experimenter contact) showed significantly less improvement on the similarities and vocabulary tasks than the training groups, suggesting that a certain amount of upward generalization occurred with the training. At a more general level, there are reports that intellectually brighter patients are better at independent living and occupational functioning than those of lower intelligence (60). The challenge is to move to a more specific realm and identify the particular cognitive deficits that become "rate-limiting factors." Just as a rate-limiting factor restricts the flow of a chemical equation, certain cognitive deficits can serve as limiting factors for effective social and occupational functioning. Other cognitive deficits may turn out to be relatively unimportant to adaptive functioning.

Several recent studies, using both cross-sectional and longitudinal designs, have searched for rate-limiting cognitive factors in social skills acquisition and social competency. Mueser et al. (61) found that baseline measures of verbal memory (but not symptoms) predicted improvement in social skills in a 2-week skills training program. For example, a summary memory score correlated 0.51 with the amount of improvement in social skills at a 1-month follow-up assessment. In a rare convergence of findings, verbal memory and susceptibility to distraction have emerged as strong predictors of both shorter (1-day) (62) and much longer (8-month) (63) periods of skills training. The findings have a certain face validity because the skills training programs place demands on processing of verbal information. These studies used relatively chronic patients, so generalizability to other patient groups remains an open question. Of course, simply finding correlations between skills acquisition and information-processing variables does not logically guarantee that these deficits are rate-limiting factors. The litmus paper test (to use another chemistry analogy) would be remediation of the more elementary information-processing deficit and then determination of whether improved skills acquisition follows.

Another type of upward generalization would be from the cognitive to the symptomatic level. Such a notion has received some empirical support from Brenner et al. (30), who examined the effects of cognitive treatments and skills training on psychotic symptoms. Their cognitive training module is directed at processes that are relatively complex compared with the elementary functions assessed by most vulnerability studies. Nonetheless, Brenner et al. reported both improved cognitive performance and symptom reduction resulting from their intervention. Although one may question whether the effects of the Brenner et al. program derive from nonspecific factors (e.g., therapist contact) (64), the integrated approach of these researchers is commendable.

Overall, the findings on the amount of generalization between cognitive levels appear to be mixed. However, it should be stressed that very little work has been conducted on this question. To address this issue effectively, a design is required in which a cognitive interven-

tion directed at a complex level is compared with an intervention directed at an elementary level. To my knowledge, no such comparison has been conducted.

The pattern of results across the few empirical studies in this area raises an intriguing (and very preliminary) suggestion. Perhaps the cognitive deficits that contribute to onset of psychosis (and presumably also lead to subsequent relapses) are different from the deficits that restrict the social and occupational functioning of the patients. If so, cognitive remediation would have to be tailored to the situation at hand. Spaulding and Sullivan (65) have suggested that remediation programs should be suited to an individual's cognitive profile. Similarly, remediation efforts might eventually be keyed to the phase of the illness. For a child who is at risk for schizophrenia, cognitive remediation might target putative vulnerability indicators in an effort to reduce the likelihood of onset of the disorder. For an adult in a psychotic episode, cognitive remediation might focus on the putative episode indicators, especially if there is reason to believe that the episode indicators contributed to the relapse. For an adult in partial remission who is trying to maintain a job, more complex skills such as verbal memory and freedom from distractibility might be targeted in a remediation program. The apparent discrepancy between putative vulnerability indicators (e.g., perceptual measures) and putative rate-limiting factors (e.g., verbal memory), although provocative, might be due to trivial factors. We tend to find only what we are looking for, and verbal memory may not have received as much investigation in the literature on high risk and vulnerability.

RECOMMENDATIONS

Substantial efforts have been directed at identifying and conceptualizing information-processing deficits in the psychoses. The gains in this field have been impressive and have made meaningful contributions to our understanding of the etiology and course of the disorders. This article considers whether it is time to move beyond identifying these deficits to the remediation of them. This move is far from a simple matter, and it requires some thought about theoretical frameworks and the basis for selecting certain deficits, among many, for remediation. I would argue that the literature (albeit sparse) and the benefits (potentially large) can currently justify small-scale, focused investigations into cognitive remediation.

Such focused investigations could serve to justify, direct, and refine more comprehensive cognitive remediation programs that are largely modeled on the types of programs available for patients with head injuries. Investigators such as Spaulding (65), Jaeger (66), Yozawitz (67), and their colleagues deserve credit for establishing such programs and helping to apply these techniques to groups of psychiatric patients.

If modest studies are justified, what types of designs would be appropriate for the questions raised in this

commentary? I tentatively offer a few suggestions, which are intended only as catalysts for discussion.

Search for Rate-Limiting Factors

Perhaps the prerequisite challenge for cognitive remediation in psychoses is to determine which cognitive deficits in fact restrict the functioning of patients (68). The field would benefit from correlational studies that assess a variety of information-processing abilities (either elementary functions or intermediate functions such as social schema) and then determine their relation to outcome functions such as social skills acquisition, social competency, or occupational functioning. Examples of this type of approach include the studies of memory and social skills acquisition (61, 63). Cross-sectional studies of this type would be informative, although longitudinal studies would be more meaningful regarding the predictive validity of deficits in elementary processes. Exploration of rate-limiting factors would involve two stages. First, in a hypothesis-generating phase, an observed association between relatively elementary cognitive deficits and a reduction in a more global outcome measure would suggest that a rate-limiting factor is operating. Second, in a hypothesis-testing approach, attempts could be made to remedy the elementary deficit and determine whether ratings on the global measure improve.

Feasibility Studies

Once relevant deficits have been identified, the next challenge is to determine whether the deficits are modifiable. It would be highly informative to use a between-subjects design to pit one type of intervention against another. For example, one group of subjects who receive general stimulation (repeated practice) could be contrasted with a group who receive a behavioral intervention (monetary reinforcement), a group who receive strategy training (e.g., instructions), and a group who receive both forms of intervention. Hence, the separate and additive effects of monetary reinforcement and instructional training could be assessed. Such an approach would help address the question of whether the effects are specific to the intervention or attributable to nonspecific factors such as experimenter contact (64). This type of study allows us to evaluate the modifiability of the cognitive deficits and to determine the relative efficacy of different types of intervention. There is no reason to suspect that one type of intervention would be equally effective for all information-processing tasks. If short-term improvement is found, questions of stability of the effect over time would need to be addressed through follow-up testings.

Studies of Generalization

When cognitive remediation is criticized, it is usually in relation to the question of generalizability of effects. After all, the eventual challenge for any intervention

will be to improve the quality of life for the patients. Generalization issues could be addressed broadly by considering both upward and downward generalization. For example, a between-subjects design could be used in which each group receives a single intervention, but some groups receive training on rather elementary processes (such as perception, attention, or memory) and other groups receive training on more global processes (such as communication skills, emotion recognition, or social schema). A standard battery of outcome measures across a variety of domains could determine the extent to which training at one level generalizes, either upward or downward, to another level. Generalization issues would be addressed in a more meaningful way with outpatients, for whom the outcome measures could include occupational and functional indexes.

If an intervention should be found to generalize according to meaningful indexes, what of the issue of cost-effectiveness? Braff has argued cogently that "it is the relative cost-effectiveness of antipsychotic versus psychosocial treatment that has fueled pessimism regarding the relatively labor-intensive cognitive remediation strategies" (69, p. 37). One response to the cost-benefit issue is to consider the relevant outcome measure. Although use of neuroleptics is certainly the most cost-effective manner of reducing symptoms, it may not be as helpful in increasing occupational and social functioning (70, 71). It is recognized that for many patients, it is easier to reduce symptoms than it is to return them to work. Such a discrepancy in outcomes (which is partly the result of premorbid factors) is also likely due to the lingering cognitive restrictions, for which cognitive remediation might eventually offer an advantage.

CONCLUSIONS

In summary, the area of cognitive remediation with schizophrenic patients suffers from a double liability: sparse data and limited theory. The existing literature is modest in size, but it suggests that some cognitive deficits are remediable, in the narrow sense that performance on a given task can often be improved. Moreover, the cognitive deficits that underlie poor performance probably restrain acquisition of information, as well as social and occupational functioning. As more effective pharmacological agents become available, it is likely that cognitive limitations will become increasingly important, because clinicians will still need to address enduring cognitive deficits, which often linger long after symptoms subside (70, 71).

We all hope that new technologies will improve the quality of life for our patients. However, I would argue that the existing database currently justifies neither optimism nor pessimism regarding the eventual value or role of cognitive remediation in psychotic disorders. In contrast, we can be highly enthusiastic about the testability of the questions in this area. The issues and constructs in this commentary lend themselves to operational definitions, and the questions lend themselves to

scientific evaluation. I have included modest suggestions for designs with the intention of providing points of departure for investigators. Although the outcome of the endeavor is unpredictable, the questions in this area invite exploration.

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The Concept of Boundaries in Clinical Practice: Theoretical and Risk-Management Dimensions

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The authors systematically examine the concept of boundaries and boundary violations in clinical practice, particularly as they relate to recent sexual misconduct litigation. They selectively review the literature on the subject and identify critical areas that require explication in terms of harmful versus nonharmful boundary issues short of sexual misconduct. These areas include role; time; place and space; money; gifts, services, and related matters; clothing; language; self-disclosure and related matters; and physical contact. While broad guidelines are helpful, the specific impact of a particular boundary crossing can only be assessed by careful attention to the clinical context. Heightened awareness of the concepts of boundaries, boundary crossings, and boundary violations will both improve patient care and contribute to effective risk management.

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Role boundaries may be crisp or flexible or fuzzy, depending on the role under consideration and on the cultural climate.

—Ingram (1)

The concept of boundaries, particularly in the sense of boundary crossings and boundary violations, has come under increased scrutiny in relation to the wave of sexual misconduct cases (2) arising in litigation, ethics committee hearings, and complaints to boards of licensure. Like many concepts in psychotherapy, such as “therapy,” “transference,” and “alliance,” the term proves slippery on closer observation. The literature tends to focus on patient-therapist sexual misconduct (3) as an extreme violation and not on the wide variety of lesser and more complex boundary crossings, many of which are, at first glance, less obvious but pose difficulties of their own for clinicians.

Clinicians tend to feel that they understand the concept of boundaries instinctively, but using it in practice or explaining it to others is often challenging. This latter problem is rendered more difficult by the tendency of the legal system, particularly plaintiffs’ attorneys, to

apply it mechanistically: any boundary crossing is bad, wrong, and harmful. Empirical evidence suggests that boundary violations frequently accompany or precede sexual misconduct (2, 4, 5), but the violations themselves do not always constitute malpractice or misconduct or even bad technique. However, modern clinicians should be aware of three principles that govern the relationship among boundaries, boundary crossings, boundary violations, and sexual misconduct.

First, *sexual misconduct usually begins with relatively minor boundary violations*, which often show a crescendo pattern of increasing intrusion into the patient’s space that culminates in sexual contact. A direct shift from talking to intercourse is quite rare; the “slippery slope” is the characteristic scenario. As Gabbard (4) and Simon (6) have pointed out, a common sequence involves a transition from last-name to first-name basis; then personal conversation intruding on the clinical work; then some body contact (e.g., pats on the shoulder, massages, progressing to hugs); then trips outside the office; then sessions during lunch, sometimes with alcoholic beverages; then dinner; then movies or other social events; and finally sexual intercourse.

Second, *not all boundary crossings or even boundary violations lead to or represent evidence of sexual misconduct*. A clear boundary violation from one ideological perspective may be standard professional practice from another. For example, the so-called “Christian psychiatry movement” might condone the therapist’s attendance at a church service with one or more patients, and various group therapeutic approaches or therapeutic communities may involve inherent boundary violations, as when some behaviorist schools permit hiring patients in therapy to do work in the treatment

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setting. Bad training, sloppy practice, lapses of judgment, idiosyncratic treatment philosophies, regional variations, and social and cultural conditioning may all be reflected in behavior that violates boundaries but that may not necessarily lead to sexual misconduct, be harmful, or deviate from the relevant standard of care.

Third, despite this complexity, *fact finders*—civil or criminal juries, judges, ethics committees of professional organizations, or state licensing boards—often believe that the presence of boundary violations (or even crossings) is presumptive evidence of, or corroborates allegations of, sexual misconduct.

To summarize the foregoing more concisely, albeit metaphorically, smoke usually leads to fire; one can, however, find smoke where there is no fire, and yet fact finders may assume that where there's smoke, there's fire. This metaphor is not trivial. In a notorious Massachusetts case (in which the doctor accused of sexual misconduct was eventually exonerated), the Board of Registration in Medicine, the state licensing authority, noted in the course of the process, "There was an undisputed level of intimacy between the two [patient and doctor] that supports the inference of sexual relations" (transcript of board proceedings, citation withheld). In its language here, the board clearly articulated its "inference" of fire from the "undisputed" presence of smoke. Moreover, recent court decisions suggest a trend toward findings of liability for boundary violations even in the absence of sexual contact (7). On this basis, the risk-management value of avoiding even the appearance of boundary violations should be self-evident.

This communication has three goals: 1) to review the subject in order to define, describe, and illustrate the range of boundary issues, 2) to demonstrate that crossing certain boundaries may at times be salutary, at times neutral, and at times harmful, and 3) to suggest preventive and reparative measures for clinicians dealing with boundary violations in themselves and their patients.

DEFINITIONS

What is a boundary? Is it too amorphous, protean, and abstract to define at all? Should we take refuge by saying, as St. Augustine was supposed to have said about time, "Time? I know what time is, provided you do not ask me"?

Part of the difficulty encountered in defining appropriate boundaries can be related to the historical tradition that modern therapists have inherited. The great figures in the field gave out mixed messages on the issue. Freud, for example, used metaphors involving the opacity of a mirror and the dispassionate objectivity of a surgeon to describe the analyst's role, but his own behavior in the analytic setting did not necessarily reflect the abstinence and anonymity that he advocated in his writings. He sent patients postcards, lent them books, gave them gifts, corrected them when they spoke in a misinformed manner about his family members,

provided them with extensive financial support in some cases, and on at least one occasion gave a patient a meal (8). Moreover, the line between professional and personal relationships in Freud's analytic practice was difficult to pinpoint. During vacations he would analyze Ferenczi while walking through the countryside. In one of his letters to Ferenczi, which were often addressed "Dear Son," he indicated that during his holiday he planned to analyze him in two sessions a day but also invited him to share at least one meal with him each day (unpublished manuscript by A. Hoffer). For Freud the analytic relationship could be circumscribed by the time boundaries of the analytic sessions, and other relationships were possible outside the analytic hours. The most striking illustration of this conception of boundaries is Freud's analysis of his own daughter, Anna.

Freud was not alone in establishing ambiguous analytic boundaries. When Melanie Klein was analyzing Clifford Scott, she encouraged him to follow her to the Black Forest for her holiday. Each day during this vacation, Scott underwent analysis for a 2-hour session while reclining on Klein's bed in her hotel room (9). D.W. Winnicott, another therapist of considerable stature, occasionally took young patients into his home as part of his treatment of them (10). In Margaret Little's report of her analysis with Winnicott (11), she recalled how Winnicott held her hands clasped between his through many hours as she lay on the couch in a near-psychotic state. On one occasion he told her about another patient of his who had committed suicide and went into considerable detail about his countertransference reactions to the patient. He also ended each session with coffee and biscuits. These boundary transgressions by highly revered figures have occasionally been cited in ethics hearings as justification for unethical behavior. We wish to stress that these behaviors are no longer acceptable practice regardless of their place in the history of our field.

The problem of the contradiction between what the master therapists wrote and how they actually behaved in the clinical setting was compounded because psychoanalysis and psychotherapy are treatments that occur in a highly private context. The boundaries of the therapeutic relationship and the characteristics of acceptable technique were thus highly subjective and lacked standardization. This lack of clarity was partially addressed by Eissler's classic 1953 paper (12) in which he suggested that in the ideal situation, the analyst's activity should be confined to interpretation. Any deviation from that model technique was defined by Eissler as a parameter. As examples of parameters, he cited Freud's setting a termination date for the Wolf Man and proposed a hypothetical situation in which an analyst might command a phobic patient to expose himself to the feared situation. By this standard of technique, Freud's own behavior, such as offering a meal to the Rat Man, has been regarded as indicative of an earlier technique that Freud subsequently abandoned (13) or a human failing rather than a technical recommendation (14).

Lipton (8) took a strikingly different view of Freud's apparently unorthodox behavior with the Rat Man. He insisted that Freud's providing a meal for the patient should not be considered part of his psychoanalytic technique. Instead, it should be regarded as part of the nontechnical personal relationship that Freud had with this patient. He pointed out that in every analysis, the analyst is called upon to offer assistance in a personal way from time to time. While the ramifications and fantasies produced by such behavior should be thoroughly analyzed, it would be erroneous, in Lipton's view, to expand the concept of technique to include all aspects of the analyst's relationship with the patient. Lipton expressed the following concern: "Modern technique tends to move from the position from which the analyst's technique is judged according to his purpose to one from which the analyst's technique is judged according to his behavior" (8, p. 262). He pointed out that following Eissler's model of analytic technique in its literal terms would cause any noninterpretive comment or action on the part of the analyst to be construed as a parameter.

Another major problem with any attempt to derive definitions of boundaries from psychoanalytic concepts of technique is that technique changes with treatments that are less expressive than analysis. As one moves along the expressive-supportive continuum of psychotherapy (15), one relies less on interpretation and more on alternative interventions such as clarification, confrontation, advice and praise, suggestion, and affirmation. Similarly, partial gratification of transference wishes is associated with supportive psychotherapy, whereas it is generally eschewed in psychoanalysis or highly expressive psychotherapy. Hence, there may be a built-in confusion between the notion of therapeutic boundaries and adjusting the technique to the ego organization of the patient.

Another approach to defining therapeutic boundaries is to conceptualize a therapeutic frame (16, 17), i.e., an envelope or membrane around the therapeutic role that defines the characteristics of the therapeutic relationship. The analyst or therapist constructs the elements of the frame partly consciously and partly unconsciously. These elements include the regular scheduling of appointments, the duration of the appointments, arrangements for payment of the fee, and the office setting itself.

Does the patient's role have a boundary? Spruiell (17) has noted that although the frame is deliberately unbalanced, the patient invariably joins the analyst in elaborating the frame. Most clinicians would agree, basing this answer on recollected violations they have witnessed, such as the patient who refers to the therapist as "Shrinkie" or springs from the chair and tries without warning to sit on the therapist's lap. It is clear, however, that the patient's boundary is a more forgiving and flexible one. The patient cannot be stopped from calling the therapist names, and that is part of the therapeutic process. The patient can be late and that can be discussed, but the therapist should not be late,

and so on. In any case our focus here is on the clinician's boundary.

Let us also agree that the role of therapist embraces the structural aspects of therapy in addition to the content; these include time, place, and money, which may, together with other aspects discussed below, represent possible sites for boundary crossings or violations to occur. If this exploration is to be useful, we should adopt the convention that "boundary crossing" in this article is a descriptive term, neither laudatory nor pejorative. An assessor could then determine the impact of a boundary crossing on a case-by-case basis that takes into account the context and situation-specific facts, such as the possible harmfulness of this crossing to this patient. A violation, then, represents a *harmful* crossing, a transgression, of a boundary. An example might be the case of a patient who had experienced severe or traumatic boundary violations in childhood and who might consequently be highly sensitive to later violations, even those usually considered benign. Note also that the difference between a harmful and a nonharmful boundary crossing may lie in whether it is discussed or discussable; clinical exploration of a violation often defuses its potential for harm.

To organize the discussion we consider the matter of boundaries, boundary crossings, and boundary violations under a series of headings: role; time; place and space; money; gifts, services, and related matters; clothing; language; self-disclosure and related matters; and physical contact. Sexual misconduct as an extreme boundary violation has been extensively addressed elsewhere (2, 4, 6, 18) and is not separately reviewed here. We should also point out that in addition to serving as antecedents to sexual misconduct, some of these areas of boundary crossing may represent ethical violations in and of themselves.

ROLE

Role boundaries constitute the essential boundary issue. To conceptualize this entity, one might ask, "Is this what a therapist does?" Although subject to ideological variations, this touchstone question not only identifies the question of clinical role but serves as a useful orienting device for avoiding the pitfalls of role violations.

A middle-aged borderline patient, attempting to convey how deeply distressed she felt about her situation, leaped from her chair in the therapist's office and threw herself to her knees at the therapist's feet, clasping his hand in both of her own and crying, "Do you understand how awful it's been for me?" The therapist said gently, "You know, this is really interesting, what's happening here—but it isn't therapy; please go back to your chair." The patient did so, and the incident was explored verbally.

Although such limit setting may appear brusque to some clinicians, it may be the only appropriate response to halt boundary-violating "acting in" (especially of the

impulsive or precipitous kind) and to make the behavior available for analysis as part of the therapy.

Almost all patients who enter into a psychotherapeutic process struggle with the unconscious wish to view the therapist as the ideal parent who, unlike the real parents, will gratify all their childhood wishes (19). As a result of the longings stirred up by the basic transference situation of psychotherapy or psychoanalysis, it is imperative that some degree of abstinence be maintained (20). However, strict abstinence is neither desirable nor possible, and total frustration of all the patient's wishes creates a powerful influence on the patient in its own right (8, 19).

In attempting to delineate the appropriate role for the therapist vis-à-vis the patient's wishes and longings to be loved and held, it is useful to differentiate between "libidinal demands," which cannot be gratified without entering into ethical transgressions and damaging enactments, and "growth needs," which prevent growth if not gratified to some extent (21). Greenson (22) made a similar distinction when he noted that the rule of abstinence was constructed to avoid the gratification of a patient's neurotic and infantile wishes, not to lead to a sterile form of treatment in which all the patient's wishes are frustrated.

Efforts to delineate the two varieties of needs often lead to problems in the area of defining the appropriate role for the therapist. Certainly, the patient may have legitimate wishes to be empathically understood, but when the therapist goes too far in the direction of trying to provide parental functions that were not supplied by the original parents, the patient may experience the therapist as making false promises. Casement (21) expressed reservations about Freud's providing a meal to the Rat Man because of the possibility that the patient may have experienced Freud's taking responsibility for a particular part of his life as an implicit promise that Freud was prepared to take over responsibility for other areas of the patient's life as well. Clearly, a therapist cannot become the "good mother" or "good father" in a literal sense and attempt to make up for all the deprivations of childhood. Even when therapists feel as though they are being coerced into a parental role by their patients, they must strive not to conform to the patients' expectations. Spruiell (17) made the following observation: "It is as disastrous for analysts to actually treat their patients like children as it is for analysts to treat their own children as patients" (p. 12).

The therapist's role is subject to some variation, of course. While most therapy is talking, there may be times when it is appropriate, for example, to write a letter on a patient's behalf. Under some circumstances, such a "breach of the frame" (16) might constitute a boundary violation, as when the therapist attempts to intervene in some extratherapeutic realm of the patient's life (e.g., a therapist wrote a stern letter to a patient's employer rebuking the latter for giving the patient excessively burdensome tasks on the job). In addition, since different modalities of therapy are commonly combined, the "talking therapist" might appro-

priately give medications, conceivably by injection at times—a clear boundary crossing but presumably therapeutic and benign.

TIME

Time is, of course, a boundary, defining the limits of the session itself while providing structure and even containment for many patients, who derive reassurance because they will have to experience the various stresses of reminiscing, reliving, and so forth for a set time only. The beginnings and endings of sessions—starting or stopping late or early—are both susceptible to crossings of this boundary. Such crossings may be subtle or stark.

A male psychiatrist came in to the hospital to see his female inpatient for marathon sessions at odd times, such as from 2:00 to 6:00 in the morning, rationalizing that this procedure was dictated by scheduling problems. This relationship eventually became overtly sexual.

An interesting prejudice about violating the boundary of time has evolved in sexual misconduct cases, a prejudice deriving from the fact that a clinician interested in having a sexual relationship with a patient might well schedule that patient for the last hour of the day (although, of course, after-work time slots have always been popular). In the fog of uncertainty surrounding sexual misconduct (usually a conflict of credibilities without witnesses), this factor has gleamed with so illusory a brightness that some attorneys seem to presume that because the patient had the last appointment of the day, sexual misconduct occurred! Short of seeing patients straight through the night, this problem does not seem to have a clear solution. Admittedly, however, from a risk-management standpoint, a patient in the midst of an intense erotic transference to the therapist might best be seen, when possible, during high-traffic times when other people (e.g., secretaries, receptionists, and even other patients) are around.

Langs (23) noted that the boundary of time may be psychologically violated when the therapist brings up material from a previous session. Some patients, indeed, feel that this practice is disruptive and is a departure by the therapist from the here and now. However, most clinicians would regard this view as extreme, since effective therapy depends on continuity from session to session.

The issue of the appropriateness of phone calls between psychotherapy sessions is a controversial one, particularly when the patient suffers from borderline personality disorder. Some therapists view such phone calls as necessary and expectable in light of the borderline patient's difficulties with evocative memory (24, 25). In other words, the patient's inability to evoke a holding, soothing introject causes anxiety of catastrophic proportions related to the fear that the therapist has disappeared. Phone calls are a way of reestablishing contact with the therapist and soothing this anxiety, which might otherwise lead to ill-advised self-

destructive behavior. On the other hand, other therapists view such calls as unnecessary and countertherapeutic (26, 27). These therapists go to great lengths in the initial contractual period, at the beginning of therapy, to extract an agreement from the patient that phone calls will be used only in emergency situations. This controversy reflects how a boundary violation may be defined according to the extent to which the appropriate treatment is viewed as having an expressive versus a supportive emphasis.

PLACE AND SPACE

The therapist's office or a room on a hospital unit is obviously the locale for almost all therapy; some exceptions are noted in the next section. Exceptions usually constitute boundary crossings but are not always harmful. Some examples include accompanying a patient to court for a hearing, visiting a patient at home, and seeing a patient in the intensive care unit after an overdose or in jail after an arrest.

Some boundary crossings of place can have a constructive effect. As with medication, the timing and dosage are critical.

After initially agreeing to attend his analysand's wedding, the analyst later declined, reasoning that his presence would be inappropriately distracting. Later, after the death of the analysand's first child, he attended the funeral service. Both his absence at the first occasion and his presence at the second were felt as helpful and supportive by the analysand. They both agreed later that the initial plan to attend the wedding was an error.

A relevant lesson from this example is that boundary violations can be reversed or undone with further consideration and discussion. At times, an apology by the therapist is appropriate and even necessary.

Some sexual misconduct cases reveal space violations that seem to manifest wishes for fusion on the part of the therapist, as in the following case.

A lesbian therapist treating a female patient would contrive to use the bathroom at the clinic whenever the patient did so and, entering the adjoining stall, would attempt to continue the conversation. The relationship became overtly and exploitatively sexual, with the therapist often wearing the patient's clothes to work the next day after they had spent the night together.

Sorties out of the office usually merit special scrutiny. While home visits were a central component of the community psychiatry movement, the shift in the professional climate is such that the modern clinician is best advised to perform this valuable service with an opposite-sex chaperon and to document the event in some detail.

Sessions during lunch are an extremely common form of boundary violation. This event appears to be a common way station along the path of increasing boundary crossings culminating in sexual misconduct. Although

clinicians often advance the claim that therapy is going on, so, inevitably, is much purely social behavior; it does not *look* like therapy, at least to a jury. Lunch sessions are not uncommonly followed by sessions during dinner, then just dinners, then other dating behavior, eventually including intercourse.

Sessions in cars represent another violation of place. Typically, the clinician gives the patient a ride home under various circumstances. Clinician and patient then park (e.g., in front of the patient's house) and finish up the presumably therapeutic conversation. From a fact finder's viewpoint, many exciting things happen in cars, but therapy is usually not one of them.

The complexity of the matter increases, however, when we consider other therapeutic ideologies. For example, it would not be a boundary violation for a behaviorist, under certain circumstances, to accompany a patient in a car, to an elevator, to an airplane, or even to a public restroom (in the treatment of paruresis, the fear of urinating in a public restroom) as part of the treatment plan for a particular phobia. The existence of a body of professional literature, a clinical rationale, and risk-benefit documentation will be useful in protecting the clinician in such a situation from misconstruction of the therapeutic efforts.

MONEY

Money is a boundary in the sense of defining the business nature of the therapeutic relationship. This is not love, it's work. Indeed, some would argue that the fee received by the therapist is the only appropriate and allowable material gratification to be derived from clinical work (28). Patient and clinician may each have conflicts about this distinction (29), but consultative experience makes clear that trouble begins precisely when the therapist stops thinking of therapy as work.

On the other hand, most clinicians learned their trade by working with indigent patients and feel that some attempt should be made to pay back this debt by seeing some patients for free—a form of “tithing,” if you will. Note that this *decision*—to see a patient for free and to discuss that with the patient—is quite different from simply letting the billing lapse or allowing the debt to mount. The latter examples are boundary crossings, perhaps violations.

Consultative experience also suggests that the usual problem underlying a patient's mounting debt is the clinician's conflict about money and its dynamic meanings. Initially reluctant to bring up the unpaid bill, the clinician may soon become too angry to discuss it. Explorations of the dynamic meaning of the bill are more convincing when they do not take place through clenched teeth. A clinician stuck at this countertransference point may simply let it slide. In the minds of fact finders, this raises a question: “The clinician seems curiously indifferent to making a living; could the patient be paying in some other currency?”—a line of speculation one does not wish to foster.

In rural areas even today, payments to physicians may take the form of barter: when the doctor delivers your child, you pay with two chickens and the new calf. For the dynamic therapist this practice poses some problems, because it blurs the boundary between payment and gift (covered in the next section). The clinician should take a case at a reasonable fee or make a *decision* to see the patient for a low fee (e.g., one dollar) or none. Barter is confusing and probably ill-advised today. Of course, all such decisions require documentation.

GIFTS, SERVICES, AND RELATED MATTERS

A client became very upset during an interview with her therapist and began to cry. The therapist, proffering a tissue, held out a hand-tooled Florentine leather case in which a pocket pack of tissues had been placed. After the patient had withdrawn a tissue, the therapist impulsively said, "Why don't you keep the case?" In subsequent supervision the therapist came to understand that this "gift" to the patient was an unconscious bribe designed to avert the anger that the therapist sensed just below the surface of the patient's sorrow.

This gift was also a boundary violation, placing unidentified obligations on the patient and constituting a form of impulsive acting in. A related boundary violation is the use of favors or services from the patient for the benefit of the therapist, as Simon's startling vignette illustrates:

Within a few months of starting . . . psychotherapy, the patient was returning the therapist's library books for him "as a favor." . . . The patient began having trouble paying her treatment bill, so she agreed—at the therapist's suggestion—to clean the therapist's office once a week in partial payment . . . The patient also agreed to get the therapist's lunch at a nearby delicatessen before each session. (6, p. 106)

The obvious exploitive nature of these boundary violations destroys even the semblance of therapy for the patient's benefit.

When Freud heard that one of his patients was planning to buy a set of his complete works, he gave the patient the set as a gift (30). Immediately following the receipt of this gift, Freud's patient found that he was unable to use his dreams productively in the analysis as he had before. Freud related this "drying up" to the gift and noted, "You will see from this what difficulties gifts in analysis always make" (p. 42).

Other boundary crossings can be relatively minor but can promote a chain of subsequent crossings, as in this example:

A patient walked into the room while her therapist was pouring coffee from a carafe. He later described how he had felt socially incapable of not offering some coffee to the patient and had indeed offered some. At the next session, the patient brought doughnuts.

As the vignette shows, many boundary problems may arise at the interface between manners and technique.

In contrast to the potentially harmful or at least confusing effects of the preceding examples, compare the practice (not uncommon among psychopharmacologists) of giving patients, as part of treatment, educational texts designed for laypersons (e.g., giving *Moodswing* [31] to a patient with bipolar disorder). Such a boundary crossing may foster mastery of the illness through information—a positive result. A similar point might be made for judicious "gifts" of medication samples for indigent patients. These two instances represent clear boundary crossings that have some justification. Ideally, even these should be discussed with an ear to any possible negative effects.

A patient in long-term therapy had struggled for years with apparent infertility and eventually, with great difficulty, arranged for adoption of a child. Two years later she unexpectedly conceived and finally gave birth. Her therapist, appreciating the power and meaning of this event, sent congratulatory flowers to the hospital.

In this case, the therapist followed social convention in a way that—though technically a boundary crossing—represented a response appropriate to the real relationship. Offering a tissue to a crying patient and expressing condolences to a bereaved one are similar examples of appropriate responses outside the classic boundaries of the therapeutic relationship.

CLOTHING

Clothing represents a social boundary the transgression of which is usually inappropriate to the therapeutic situation, yet a patient may appropriately be asked to roll up a sleeve to permit measurement of blood pressure. Excessively revealing or frankly seductive clothing worn by the therapist may represent a boundary violation with potentially harmful effects to patients, but the issue can also be overdone, as in the following case.

A patient in a western state, as part of a sexual misconduct allegation that a jury later found to be false, accused the therapist (among other things) of conducting therapy sessions with the top two buttons of his shirt undone. While such a phenomenon might conceivably represent a violation for a very sensitive patient, evidence was introduced that revealed the exaggerated nature of this claim in *this* case.

Berne (32) noted the technical error of the male clinician who, confronting a patient whose skirt was pulled up high, began to explain to the patient his sexual fantasies in response to this event. Berne suggested instead saying to the patient, "Pull your skirt down." Similar directness of limit setting appears to be suited to the patient who—either from psychosis or the wish to provoke—begins to take off her clothes in the office. As before, the comment, "This behavior is inappropriate,

and it isn't therapy; please put your clothes back on," said in a calm voice, is a reasonable response.

LANGUAGE

As part of the otherwise laudable efforts to humanize and demystify psychiatry a few decades back, the use of a patient's first name was very much in vogue. While this may indeed convey greater warmth and closeness, such usage is a two-edged sword. There is always the possibility that patients may experience the use of first names as misrepresenting the professional relationship as a social friendship (28). There may well be instances when using first names is appropriate, but therapists must carefully consider whether they are creating a false sense of intimacy that may subsequently backfire.

A middle-aged woman tried for more than a year to get her therapist to use her first name, but the requests were denied, and exploration of the issue took place instead. After some time the patient recovered memories of previously repressed material, in part because of increased trust in the therapist. The patient spontaneously related her trust to the use of last names as a boundary issue; boundaries had been badly blurred in her family and this had included sexual abuse.

There are distinct advantages to addressing the adult in the patient, in terms of fostering the adult observing ego for the alliance. Trainees often do not see the paradox of expecting adult behavior on the ward from someone they themselves call "Jimmy," which is what people called the patient when he was much younger. Last names also emphasize that this process is work or business, an atmosphere which may promote a valuable mature perspective and minimize acting out. In addition, calling someone by the name used by primary objects may foster transference perceptions of the therapist when they are not desirable, as with a borderline patient prone to forming severe psychotic transferences. For balance, however, recall that use of last names may also sound excessively distant, formal, and aloof.

Tone is also a part of language. A patient won a settlement in an allegation of sexual misconduct when the tape recording she had made of a phone call from her therapist revealed his intimate, seductive tone. The therapist's attorney urged the settlement for fear that the jury would hear the intimate tone as evidence of a sexual relationship.

Word choice can also be violative, as when the therapist inquires, "What are you feeling now in your vagina?" Note that this inquiry might be proper in analytic therapy after appropriate preparation. Clinical utility aside, the way in which such explorations may violate boundaries should be kept in mind.

Finally, psychotherapy may be a forum for sado-masochistic enactments in which aggressive verbal abuse grows out of countertransference sadism. Cruel and contemptuous comments by the therapist may be rationalized as therapeutic confrontation.

SELF-DISCLOSURE AND RELATED MATTERS

Few clinicians would argue that the therapist's self-disclosure is always a boundary crossing. Psychoanalysis and intensive psychotherapy involve intense personal relationships. A useful therapeutic alliance may be forged by the therapist's willingness to acknowledge that a painful experience of the patient is familiar to himself (19). However, when a therapist begins to indulge in even mild forms of self-disclosure, it is an indication for careful self-scrutiny regarding the motivations for departure from the usual therapeutic stance. Gorkin (33) observed that many therapists harbor a wish to be known by their patients as a "real person," especially as the termination of the therapy approaches. While it may be technically correct for a therapist to become more spontaneous at the end of the therapeutic process, therapists who become more self-disclosing as the therapy ends must be sure that their reasons for doing so are not related to their own unfulfilled needs in their private lives but, rather, are based on an objective assessment that increased focus on the real relationship is useful for the patient in the termination process.

Self-disclosure, however, represents a complex issue. Clearly, therapists may occasionally use a neutral example from their own lives to illustrate a point. Sharing the impact of a borderline patient's behavior on the therapist may also be useful. The therapist's self-revelation, however, of personal fantasies or dreams; of social, sexual, or financial details; of specific vacation plans; or of expected births or deaths in the family is usually burdening the patient with information, whereas it is the patient's fantasies that might best be explored. The issue is somewhat controversial: a number of patients (and, surprisingly, some therapists) believe that the patient is somehow entitled to this kind of information. In any case, it is a boundary violation and as such may be used by the legal system to advance or support a claim of sexual misconduct. The reasoning is that the patient knows so much about the therapist's personal life that they must have been intimate (compare the remark by a board of registration, quoted earlier).

Subtler variations on the information theme may occur, as when a therapist sees members of a couple in parallel treatment but separately alludes in one member's session to material from the other's. Sensitivity in this area may run quite high.

A patient had a dream involving Nazis. In the interpretation the therapist suggested that this detail referred to himself. The patient seemed doubtful. The therapist noted that the interpretation was based in part on the fact that other patients of his had dreamed of Nazis in response to the therapist's German last name. The patient's mood changed; only later was she able to tell the therapist how violated she had felt at his "intruding" other patients into the session.

Although the intrusion was at a verbal level only, the impact was clear for this patient, who had been sub-

jected to some disregard of her boundaries in previous therapy.

Finally, the boundary can be violated from the other side. An example would be the therapist's using data from the therapy session for personal gain, such as insider information on stock trading, huge profits to be made in real estate, and the like.

PHYSICAL CONTACT

To place the issue of physical contact in context, it should be noted that psychiatrists traditionally performed their own physical examinations. This practice has declined so markedly that a senior psychiatrist recently wrote about examining a patient's bruised leg as a major return to the past. Hospitals commonly use internists for this purpose. Psychiatric residents still do their own physical examinations but commonly maintain distance by examining each other's patients. Abnormal Involuntary Movement Scale examinations for tardive dyskinesia are often the only routine physical contact.

There is room here for regrets. Physicians working with a patient with AIDS or HIV seropositivity often describe wishing to touch the patient in some benign manner (pat the back, squeeze an arm, pat a hand) in every session. They reason that such patients feel like lepers, and therapeutic touch is called for in these cases. But even such humane interventions must be scrutinized and, indeed, be documented to prevent their misconstruction in today's climate.

From the viewpoint of current risk-management principles, a handshake is about the limit of social physical contact at this time. Of course, a patient who attempts a hug in the last session after 7 years of intense, intensive, and successful therapy should probably not be hurled across the room. However, most hugs from patients should be discouraged in tactful, gentle ways by words, body language, positioning, and so forth. Patients who deliberately or provocatively throw their arms around the therapist despite repeated efforts at discouragement should be stopped. An appropriate response is to step back, catch both wrists in your hands, cross the patient's wrists in front of you, so that the crossed arms form a barrier between bodies, and say firmly, "Therapy is a talking relationship; please sit down so we can discuss your not doing this any more." If the work degenerates into grabbing, consider seriously termination and referral, perhaps to a therapist of a different gender.

What is one to make of the brands of therapy that include physical contact, such as Rolfing? Presumably, the boundary extends to that limited physical contact, and the patient expects it and grants consent; thus, no actual violation occurs. Massage therapists may struggle with similar issues, however. In other ideologies the issue may again be the impact of the appearance of a violation:

A therapist—who claimed that her school of practice involved hugging her female patient at the beginning and end

of every session, without apparent harm—eventually had to terminate therapy with the patient for noncompliance with the therapeutic plan. The enraged patient filed a sexual misconduct claim against the therapist. Despite the evidence showing that this claim was probably false (a specious suit triggered by rage at the therapist), the insurer settled because of the likelihood that a jury would not accept the principle of "hug at the start and hug at the end but no hugs in between." If the claim was indeed false, this is a settlement based on boundary violations alone.

At another level this vignette nicely suggests how nonsexual boundary violations may be harmful to a patient in much the same way that actual sexual misconduct is. Instead of engaging the patient in a mourning process to deal with the resentment and grief about the deprivations of her childhood, the therapist who hugs a patient is often attempting to provide the physical contact normally offered by a parent. The patient then feels entitled to more demonstrations of caring and assumes that if gratification in the form of hugs is available, other wishes will be granted as well (compare Smith's concept of the "golden fantasy" that all needs will be met by therapy [34]). When actual physical contact occurs, the crucial psychotherapeutic distinction between the symbolic and the concrete is lost (21), and the patient may feel that powerful infantile longings within will finally be satisfied.

CONCLUSIONS

Boundary crossings may be benign or harmful, may take many forms, and may pose problems related to both treatment and potential liability. The differences in impact may depend on whether clinical judgment has been used to make the decision, whether adequate discussion and exploration have taken place, and whether documentation adequately records the details. The complexity of the subject and the variability of results from case-by-case analysis merit empirical study. Educational materials are available through the Office of Public Affairs of the American Psychiatric Association. Heightened awareness of the concepts of boundaries, boundary crossings, and boundary violations will both improve patient care and contribute to effective risk management.

In an effort to prevent more serious boundary violations of a sexual nature, Epstein and Simon (28) have developed an exploitation index which comprises a list of questions that therapists can ask themselves about their current behavior with patients. In this manner these authors have attempted to provide an ongoing self-monitoring system. While such approaches may be useful for some clinicians, we must acknowledge that considerable personal variation exists in our field. The relationships between therapist and patient vary from one therapist to another, and there are even variations across patients in the practice of one therapist. As Lipton (8) observed, it is ultimately impossible to codify or prescribe a personal relationship between therapist and

patient in a precise manner. Perhaps the best risk management involves careful consideration of any departures from one's usual practice accompanied by careful documentation of the reasons for the departure. Finally, the value of consultation with a respected colleague should be a built-in part of every practitioner's risk-management program.

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Diagnostic Decision Making in Psychiatry

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The purpose of this article is to examine the consequences of and possible responses to uncertainty in psychiatric diagnosis. Uncertainty is inevitable because of the overlap in characteristics, or test results, between populations with and without a psychiatric disorder. As a result, there is never one correct method of identifying cases and noncases (i.e., case definition). In this paper principles of decision analysis and clinical epidemiology are used to develop a framework for thinking about the consequences of different diagnostic schema and for choosing among them. The framework illustrated here involves choosing an external validator, choosing a separator, and choosing a cutoff. This framework is applied to the problems facing three hypothetical researchers, and the consequences for their research of different diagnostic choices are explored. It is demonstrated how the relevance of research to clinicians and policy makers rests on the choice of the case definition process. The prevalent use of structured psychiatric interviews has not been accompanied by adequate attention to the problem of determining a diagnosis once the information is obtained. It is argued that more attention must be given to this process if we are to make optimal use of available resources for research and treatment.

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Despite much work over the last 25 years, an extensive literature documents imperfections in the nosologies, diagnostic criteria, and diagnostic tests that are currently in use in psychiatry (1–7). Critics argue that there is little evidence that most DSM-III-R disorders exist as discrete entities (8). Opinions vary about whether this is a result of the nature of psychiatric disorders themselves (and therefore is *not* amenable to improvement) or whether it is a result of less than optimal conceptualizations and tools (and therefore *is* amenable to improvement). Either way, psychiatrists today are left with a situation in which even the best-available diagnostic methods result in considerable overlap between normal and disordered populations in any relevant measure. Since many situations require that classifications be made, some misleading results are inevitable.

Methods for examining the consequences of false results and optimizing one's choices given uncertainty are referred to as "decision analysis" (9). These techniques were first used in the 1950s in the military to help de-

termine optimal "signal-to-noise" ratios for the detection of incoming missiles (10). Since then, the techniques have been widely used in business and are routinely taught to business students. Over the past 15 years decision analysis has been used in many areas of medicine to address clinical problems (10–13) and to address policy issues (10, 14–17). Decision analytic techniques have only recently begun to appear in the psychiatric literature. For example, papers by Mossman and Somoza (18), Murphy et al. (19), and others (20–22) addressed the choice of diagnostic tests in psychiatry; Baldessarini et al. (23) and Hsiao et al. (24) addressed general problems with the interpretation of diagnostic tests in psychiatry; Widiger et al. (25) examined the development and operation of diagnostic systems; and Zarin and Pass (26) analyzed a clinical decision facing a patient with bipolar disorder. Some of these issues have also been addressed in the child psychiatry literature (e.g., 27, 28).

Although many medical schools have incorporated some basic decision theory (especially relating to the interpretation of diagnostic tests) into their curricula, psychiatrists have been slower to appreciate that these principles apply to the interpretation of their clinical or research assessments. In addition to the occasional laboratory test, psychiatrists routinely use both formal and informal clinical interviews to assess their patients. Some of these interviews are designed to determine a diagnosis, and some are used to help choose a course of action. In decision analytic theory, a "test" is "any

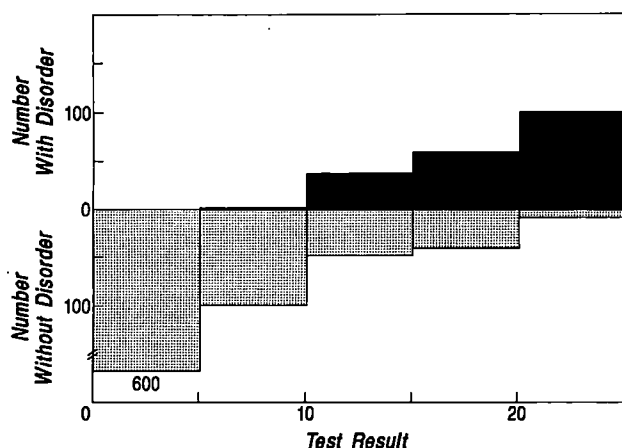
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FIGURE 1. Hypothetical Comparison of Diagnostic Test and Independent Assessment by Panel of Psychiatrists^a



^aThe 200 subjects judged by the psychiatrists to have a disorder are plotted above the x axis, and the 800 subjects judged not to have a disorder are plotted below the x axis. The vertical lines represent different cutoff values that can be used to define the diagnostic test.

means of seeking information about a patient: historical, physical, chemical, radiographic, or other" (9, p. 79). The facts that these psychiatric "tests" are subjective and at times unreliable paradoxically lead many investigators to ignore the consequences of potential errors. In fact, such considerations are at least as important in psychiatry as in fields of medicine that use more objective tests.

The types of diagnostic decisions that currently plague psychiatrists and psychiatric researchers have much in common with the therapeutic decisions for which decision analytic techniques have been useful in other areas of medicine. Specifically, they share the following features: 1) several strategies can be delineated; 2) the possible consequences of each strategy are known in general, but the specific consequence in any given instance can only be specified in a probabilistic manner; and 3) the implications (or value) of each possible outcome can be assessed. Consider, for example, a therapeutic decision that involves the treatment of a depressed person. The possible strategies might be as follows: treat with antidepressant medication alone; treat with psychotherapy and antidepressant medication; or treat with psychotherapy alone. The outcome with each strategy could be determined as the percentage of people who recover without untoward side effects within a given amount of time. The best strategy would then be the one associated, on average, with the best outcome.

A diagnostic decision, on the other hand, might involve the identification of depressed people in the community. Different strategies might involve combinations of diagnostic instruments or alternative methods of scoring them. The outcomes could include the chances of falsely identifying people through the use of each strategy. The consequences of being falsely identi-

fied could also be delineated. The best strategy would then be the one that minimized negative consequences of misclassification and/or maximized the positive consequences of correct classification.

To apply this methodology to diagnostic decisions in psychiatry, certain principles of clinical epidemiology must also be used. Clinical epidemiology is the science of applying the techniques and principles of epidemiology to the design and interpretation of clinical trials. The principles that are used in this paper all deal with the general issue of using data based on one population to inform decisions about another population. A comprehensive treatment of this subject can be found in many sources (e.g., 29).

The fields of decision analysis and clinical epidemiology have much to offer psychiatrists. This paper addresses their application to the problem of making diagnoses in a field with imperfect tests and imperfect (or at least controversial) nosologies. Whereas others have documented the existence of uncertainty in psychiatric diagnosis, we focus here on the *consequences* of that uncertainty for researchers and the consumers of their research. In addition, we present a framework that can be used to optimize diagnostic choices given the inherent uncertainty. Although these are issues of immediate concern to researchers, they affect all who depend on research to make clinical or policy decisions that affect psychiatric patients.

THE CASE DEFINITION PROBLEM

Case definition is the process by which subjects are classified as "cases" (those judged to have a disorder) or "noncases" (those judged not to have a disorder). (Throughout this paper, the terms "disordered" and "nondisordered" will refer to the true condition of the subject; "case" and "noncase" will refer to the classification that is made as a result of a diagnostic test or algorithm.) Typically, a diagnostic test is performed and the results are used to determine "caseness." The data in figure 1 demonstrate the difficulties that are commonly encountered in this task. These fictional data show the results of a psychiatric test when they are compared to an independent assessment by a panel of psychiatrists. The 200 subjects judged by the psychiatrists to have a disorder are plotted above the x axis, and the 800 subjects judged not to have a disorder are plotted below the x axis. Clearly, the higher the score on the test, the more likely it is that the person is disordered. A vertical line can be used to represent a cutoff value; the bars to the left of the line would then be judged to represent noncases, and those to the right would represent cases. A diagnostic test is defined once a cutoff is chosen; in this example, cutoffs at test scores of 5, 10, 15, and 20 would result in different tests.

It is apparent, however, that any cutoff value will yield some false results. In addition, movement of the line (i.e., the cutoff) always results in some trade-off between false positives and false negatives.

TABLE 1. Definitions of Hypothetical Diagnostic Test Based on Using Different Score Cutoffs With Same Patient Data

Test Result	Number of Subjects			Diagnostic Measure (%)			
	Disorder	No Disorder	Total	Sensitivity	Specificity	Predictive Value Positive	Predictive Value Negative
Cutoff=5				100	75	50	100
Cases	200	200	400				
Noncases	0	600	600				
Total	200	800	1,000				
Cutoff=10				95	88	66	99
Cases	190	100	290				
Noncases	10	700	710				
Total	200	800	1,000				

Typically, authors report the sensitivity and specificity for a diagnostic test. The sensitivity is the percentage of truly disordered people who are correctly identified by the test as being cases. The specificity is the percentage of truly nondisordered people who are correctly identified by the test as being noncases. Together, these two measures reveal how well the test detects disordered and nondisordered people. For any given cutoff value, the sensitivity can be determined by looking only at the disordered group (those above the x axis in figure 1); the specificity can be determined by looking only at the nondisordered group (below the axis). Clinically, however, tests are done because it is not known ahead of time which people have or do not have a disorder (although the approximate proportion of each, or prevalence, might be known). The sensitivity and specificity alone cannot help a clinician (decision maker) interpret a given test result in his or her mixed population of disordered and nondisordered people.

Two other measures are useful for clinicians: the predictive value positive and negative. The predictive value positive is the percentage of people with a positive test result (i.e., cases) who are truly disordered. The predictive value negative is the percentage of people with a negative test result (i.e., noncases) who are truly nondisordered. (Although most clinicians think about tests for individual patients, it is convenient to think about each patient as coming from a population of patients with the same characteristics. Some members of the population could be thought of as truly having the disorder in question and the others as not having the disorder. The exact mix in this theoretical population would depend on the characteristics of the index patient. The *prevalence* of disorder in the population is then mathematically equivalent to the *probability* that the individual patient has the disorder, i.e., it would range from a probability of one—certainty that the patient had the disorder—to a probability of zero—certainty that the patient did not have the disorder. Similarly, the predictive value positive and negative could be interpreted as the probability that the single patient has the disorder given a positive test result and the probability that the patient does not have the disorder given a negative result.)

Table 1 illustrates alternative interpretations of the data in figure 1. With a cutoff value of 5 on the test,

subjects whose scores are above 5 are considered cases; with a cutoff value of 10, subjects whose scores are above 10 are considered cases. The sensitivity, specificity, and predictive value positive and negative are also shown. Note that the use of different cutoff values defines different diagnostic tests with different sensitivity, specificity, etc. Although using a cutoff of 5 results in no false negatives, the test yields 200 false positive results. Using a cutoff of 10 yields 10 false negatives and 100 false positives. In fact, it is clear from figure 1 that the use of any cutoff would result in some false results. In other words, no one cutoff value is "correct."

In the preceding example, the psychiatrists' judgments were used as the standard of truth, or the "gold standard." Yet the documented lack of reliability of such judgments and unresolved questions about their validity diminish one's confidence about the value of psychiatrists' judgments. Psychiatric research has been hampered by the lack of true gold standards for the common disorders. To explore the consequences of imperfections in diagnostic tests it has thus far been assumed that there are means of determining the truth about the existence of psychiatric disorders. Later we will introduce a framework for proceeding with research in the absence of such gold standards.

The inability to perfectly differentiate between subjects with and without a given psychiatric disorder leads to problems in two separate but related areas of psychiatry: 1) the design and conduct of research and 2) clinical decision making in regard to psychiatric patients.

Design and Conduct of Research

Research that is aimed at increasing our understanding of psychiatric disorders inevitably involves case definition. The process may occur at the outset (e.g., as in a clinical trial of a new treatment) or later, in the data analysis phase (e.g., as in a population-based study of psychiatric disorders). Either way, the results will depend on the quality of the case definition strategy. We have no reason to believe that there is a perfect way of diagnosing any psychiatric disorder; thus, any group of cases is likely to include some subjects who are truly nondisordered, and any group of noncases is likely to include some subjects who are truly disordered. Research findings will vary with the particular

mix of disordered and nondisordered subjects in the case and noncase groups. In psychiatric research, for example, measures of risk factors, family history, service utilization, treatment response, and prognosis could all be profoundly affected. The effect would be to hamper our ability to make optimal clinical or policy decisions (e.g., involving reimbursement or service planning) for our patients.

Although case definition strategies cannot be empirically compared with a gold standard (since none exists), the phenomenon just described has been documented by studies that compare results based on two or more case definition strategies. These studies clearly demonstrate that findings on a given disorder, in fact, depend on the case definition strategy and therefore vary with the definition (30–33), instrument (30, 34), cutoff (32, 33, 35), and informant (30, 36–41).

Clinical Decision Making

Clinicians must make treatment decisions based on research data. Psychiatrists are accustomed to the need to consider whether their patients are similar to the patients who were studied in terms of a variety of characteristics that could affect course or treatment response (e.g., age, gender, past history). They are, perhaps, less aware of the need to control for case definition strategy, even though this variable is likely to have profound effects on the expected results of an intervention. Ideally, clinicians would use the same case definition strategies that the researchers used; only in that way would the research findings be sure to apply to the clinician's patients. Attainment of this goal is hampered by several factors: 1) the wide variety of case definition strategies in current use, 2) the poor reliability of some strategies, 3) the poor delineation of case definition strategies in many research reports, 4) the impracticality of many research strategies for clinical use, and 5) the dependence of case definition strategies on characteristics of the population, which may differ between research and clinical settings. As a result, clinicians treat a group of cases that contains a poorly defined mix of disordered and nondisordered people, and they base this treatment on research that perhaps contains a different mix. Ransohoff and Feinstein (42) showed how such case mix problems have led to overly optimistic predictions regarding the efficacies of certain diagnostic tests. Similarly, premarket testing of drugs is typically done with more carefully defined groups of patients than are treated once the drug is on the open market. This has led to a syndrome in which many new drugs are heralded as being highly effective and then are found to be substantially less effective as their clinical use broadens (43). Clearly, considerations of case mix and its effect on the clinical relevance of research are critical to optimal patient care.

The next section introduces a framework for rationally using all available diagnostic information, given imperfections in tests and disagreements about valid gold standards. To illustrate the use of this framework,

the diagnostic decisions confronting three hypothetical researchers will be explored. The examples are taken from child psychiatry, since that is the focus of most of our work. However, the concepts are equally applicable to all areas of psychiatry.

TOWARD A RATIONAL METHOD OF CASE DEFINITION

The problems described in the preceding section cannot be solved entirely. They contribute to the overall level of uncertainty in psychiatry. They are not unique to psychiatry, however. They plague all of clinical medicine, as well as numerous other fields in which decisions must be made under conditions of uncertainty. The principles of decision analysis can be used to address the case definition problem. The method involves three steps. The first step is to choose an external validator, or criterion for determining who does and does not have a disorder. The second step is to choose a separator, i.e., a diagnostic test or algorithm to distinguish the population of disordered from the nondisordered. The third step is to determine the optimal cutoff point for distinguishing positive results (cases) from negative results (noncases).

The use of this framework is illustrated by considering how three hypothetical researchers could apply it to their work:

Researcher A is interested in developing an algorithm that would enable pediatricians to identify a group of children who need psychiatric referrals because of depressive symptoms.

Researcher B is interested in evaluating the efficacies of two treatment strategies that are in current use for childhood depression. Both strategies are of minimal risk to children.

Researcher C is interested in determining whether severe and recurrent depression in children is familial.

Step 1—Choice of External Validator

The external validator is the standard against which the case definition algorithm will be compared. In other words, it determines, in theory, who does and does not have a disorder. There are many types of external validators that are potentially useful in psychiatry. In their landmark paper, Robins and Guze (3) summarized several types. These include the clinical picture, laboratory studies, longitudinal course, and family pattern. Which one of these validators is used depends on one's "construct" of the disorder (44). This has been defined as the "key defining features" of a disorder. A disorder may have several possible constructs, depending on the context. Which validator is best will depend on which constructs are deemed relevant by the research team. The construct, and therefore the choice of an external validator, depends on a value judgment: Which features of the disorder are most important to discern? The three researchers just described might consider the following.

Researcher A wants to identify children who need psychiatric evaluation for depression. However, it would presumably be a benefit if, by chance, some children were referred who needed psychiatric care for disorders other than depression. Therefore, overall assessment of disability and some assessment of the clinical picture and need for services are probably the most relevant aspects of the construct.

Researcher B is attempting to answer a question of relevance to standard clinical practice. Thus, her whole case definition strategy should reflect what a practitioner is likely to use clinically. The external validator should therefore reflect the clinician's tendency to rely on the clinical picture and degree of disability in various settings.

Researcher C is interested in studying a group of children with depression that is severe and recurrent. She should therefore choose an external validator that assesses the severity of each episode and the possibility of recurrences. Note, however, that she does not want her external validator to include a measure of family history, since this would bias her ability to test the hypothesis that severe depressions are familial.

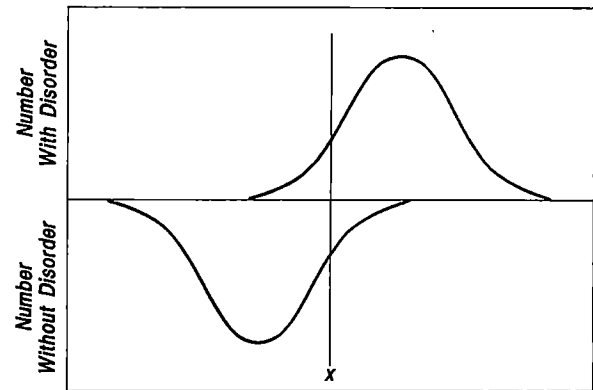
It would be ideal if different external validators identified the same groups of disordered and not disordered people, but frequently this is not the case. For example, it has been argued (44) that the DSM-III criteria do a good job of identifying schizophrenia if one values a construct based on course (i.e., it identifies psychotic patients with a chronic course and a poor prognosis) but that the criteria are too narrow if schizophrenia is primarily seen as a disorder that runs in families. Lahey et al. (33) explored the consequences of two different case definition strategies (i.e., one based on DSM-III and one based on DSM-III-R) for several disorders in prepubertal children. Not surprisingly, they found that different children were identified by the different strategies, leading to different conclusions about the disorders, depending on how they were defined. Since the choice of an external validator depends on the goals of the study, it is possible that any given study may logically use several different case definition algorithms, each one based on a different external validator.

Step 2—Choice of Separator (Discriminator)

Once an external validator is chosen, a strategy for identifying cases must be developed. Figure 2 illustrates the task. In this figure, test results for subjects who are disordered and subjects who are not disordered are plotted separately. Typically, as we have already noted, these distributions will overlap. The goal is to find the test, or separator, that minimizes the overlap between the two distributions. Many features of a diagnostic procedure affect its ability to discriminate between those with and without a disorder. Each factor is considered separately.

Instrument. The choice of instrument should relate to the external validator. For example, if the validator is psychiatric diagnosis, then an assessment procedure is

FIGURE 2. Results of a Typical Diagnostic Test Plotted Separately for Subjects With and Without Disorder^a



^aAny cutoff (here shown at point x) will result in some false results.

needed that will provide a diagnosis; this could range from an informal clinical interview to a highly structured questionnaire. (The term "instrument" will be used to cover all of these possibilities.) For most applications, many instruments are available and one (or a set of instruments) must be chosen. Typically, reliability and validity data are available to help the researcher choose among them. In the present paradigm, the important features are that the instrument reliably separate the populations by using the external validator chosen. This may or may not be consistent with the procedures used to determine the published reliability and validity.

Informant. The choice of informant is particularly difficult in studies of children but is also relevant in research on adults. For some applications, there are many possible informants and no immediately clear way to choose among them. In research on children, the possibilities frequently include the child, the parent(s), and the teacher(s). A great deal of available data (38–41) indicate that children and their parents and teachers differ in their affirmation of psychiatric symptoms in the children. For example, Kazdin (30) has shown that which children are identified as disordered depends on whether the child or the parent acts as the informant. In adult studies, the spouse and other family members are frequently considered important informants in addition to the identified subject. In other situations, the identified subject is not available for interview and a family member is interviewed instead (for instance, in family or genetic studies). Information regarding the usefulness of each possible informant is crucial to making appropriate choices.

Scoring system. The usefulness of an instrument depends also on how it is scored. Some instruments allow for a binary response (e.g., "yes" or "no") and are scored simply as the sum of positive responses. Others can be quite complicated, including those based on DSM-III-R; these instruments have criteria and subcriteria for each possible diagnosis. There are complex rules for de-

termining when a person meets the criteria for a given disorder. Even in this realm, however, there is room for variability. An instrument may allow only a yes/no response, or it may have more options, such as "always," "sometimes," or "never." Murphy et al. (19) have shown how a relatively minor change in a scoring system can affect an instrument's discriminating properties.

These complexities are magnified when one considers the possibility of combining information from different informants. When a DSM-III-R-based instrument is used and a parent and child are the informants, there are many possible scoring algorithms; some may combine information only at the diagnosis level, others may combine information at the symptom level. The combination rules define a new separator that must be considered along with the others to determine an optimal choice. Each one of the three researchers described earlier would have different priorities in choosing a separator.

Researcher A will need to use a separator that can be administered by busy pediatricians or their staff. It should be able to separate a group of children who need referral from those who do not. As mentioned, it is not quite as important that it distinguish among the psychiatric disorders which may be confused with depression but also need specialized attention. Information from parents might be considered particularly important, since they usually determine when and to whom to take their children for treatment.

Researcher B should aim to reflect what would be considered optimal clinical practice. The separator should therefore include the criteria of DSM-III-R along with some measure of disability. The information base should reflect the sources that a clinician is likely to rely on, e.g., child, parent, and perhaps teacher. The method of combining the data and the scoring system should similarly be chosen to reflect clinical practice.

Researcher C should use a separator that can distinguish a group of children with depression that is severe and recurrent. For this task, both a cross-sectional measure and a longitudinal measure are necessary. The cross-sectional measure should include items addressing a range of symptoms and a range of severities so that a more severe group can be distinguished.

Step 3—Choice of Cutoff

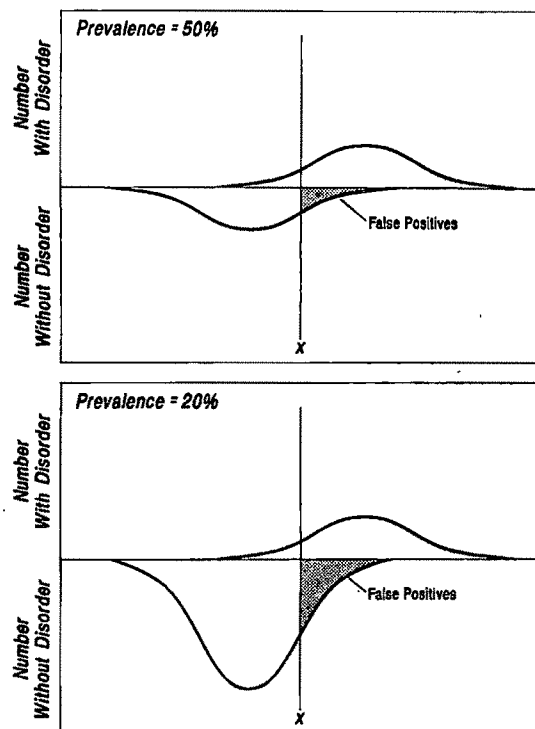
Once an external validator and a separator variable are chosen, a cutoff value, above (or below) which a subject is considered a case, must be chosen. This is sometimes called the "positivity criterion" (9). In most situations, any cutoff value chosen will result in some misclassifications. Intuitively, it seems that false positives and false negatives should be balanced in determining an optimal cutoff.

It is not enough to simply compare the absolute numbers (or percentages) of false positives and negatives, however; the consequences of these errors must also be weighed. For example, consider a test to detect an illness that is lethal unless it is treated with a drug that is

100% effective but carries minimal risk (assume also that the test itself is risk free). In this situation, the consequences of a false negative (certain death) far outweigh the consequences of a false positive (minimal risk). It would not make sense to use a cutoff that equalized the occurrence of these two errors. The rational decision maker would prefer to have more false positives to avoid excess false negatives. This example also points out that the consequences of the true results are equally important. This becomes clearest when the concepts are brought to bear on the status of an individual patient or subject. Consider a disordered person whose value on the separator variable places him or her near the likely cutoff. The question is, What are the consequences of labeling this person positive (a case) versus negative (a noncase)? Similarly, consider a nondisordered person whose score is also near the cutoff: What are the consequences of labeling him or her positive versus negative? For the disordered person, the consequences would be the difference in expected outcome between true positives and false negatives; for the nondisordered person, it would be the difference in expected outcomes between the true negatives and false positives. The important point is that the consequences of true positives, true negatives, false positives, and false negatives are all important in choosing the positivity criterion (9). (Epidemiologists distinguish between balanced and unbalanced misclassification [45, 46]. This distinction has to do with whether or not the false positives balance the false negatives. The type of misclassification, balanced or unbalanced, affects the way in which the results can be generalized from one population to another. The perspective in this paper is primarily that of the individual person. It is not relevant, therefore, to question whether one person's misclassification has been balanced by somebody else's misclassification, although this may indeed be important to determine before one draws conclusions about the consequences of the intervention, or "exposure," in question.)

Other factors are also important, however. Figure 3 demonstrates how the relative size of the disordered and nondisordered populations (i.e., the prevalence of the disorder) might affect this decision. As the nondisordered population gets relatively larger (i.e., the prevalence goes down), the number of false positives will increase. Therefore, false positives will be relatively more common in populations with a low prevalence of disorder.

The methods suggested can be used to choose a cutoff that is optimal from the point of view of someone making decisions with regard to individuals or groups of people. Research is also conducted to advance our understanding of disorders. Sackett and Gent (47) distinguished between management trials, which seek to determine whether a given treatment is likely to be effective in a given (i.e., usual) clinical situation, and explanatory trials, which seek to determine how and whether a given treatment can ever (i.e., under ideal conditions) produce a given effect. In light of this additional goal of research (i.e., as in explanatory trials), the

FIGURE 3. Results of a Typical Diagnostic Test When Applied to Populations With Different Prevalences of Disorder

predictive value positive and the predictive value negative are important. In essence, these variables tell the researcher how pure their case and noncase groups are. In other words, are the cases mainly people who are disordered (i.e., high predictive value positive) and are the noncases mainly people who are not disordered (i.e., high predictive value negative)? Methods that optimize the choice of a cutoff from a decision-making point of view do not necessarily result in high predictive values.

It is difficult to determine the predictive values for psychiatric studies. To do this, the researchers would need to publish the sensitivity and specificity of their tests or diagnostic methods. The articles that describe the Ontario Child Health Study contain such information (48, 49). This study was designed to measure the prevalence of emotional and behavioral disorders in children between the ages of 4 and 16 years. A checklist was developed and the results were compared, in a random subset of the study population, with those of a panel of child psychiatrists. The sensitivity and specificity of the checklist for each of four classes of disorder were published. From these, the predictive values could be determined. The predictive value positives ranged from 0.13 (emotional disorders, age 4–11) to 0.91 (hyperactivity, age 4–11). In other words, only 13% of the children identified as having “emotional disorder” and 91% of those identified as having hyperactivity were correctly classified. The predictive value negatives were better; they ranged from 0.86 (emotional disorder, age 4–16) to 0.99 (hyperactivity, age 12–16).

TABLE 2. Relation of Specificity and Sensitivity of Diagnostic Test to Predictive Value Positives for Population With 10% Prevalence of Disorder

Specificity	Predictive Value Positive (%)			
	Sensitivity=85%	Sensitivity=90%	Sensitivity=95%	Sensitivity=100%
85%	39	40	41	42
90%	49	50	51	52
95%	65	67	68	69
100%	100	100	100	100

Even in the absence of many published data, however, it is clear that predictive value positives are likely to be less than 70% in most epidemiological studies (since the predictive value positive depends on the prevalence, the values will be higher in clinical research, where the study population includes a high percentage of disordered subjects). Table 2 illustrates this. The predictive value positives have been computed for a test that is applied to a population with a 10% prevalence of disorder. The sensitivity and specificity of the test in detecting the disorder have been varied to illustrate their effect on the predictive value positive (this could be thought of as resulting from a change in cutoff or change in criteria). Note that the predictive value positive will be greater than 69% only when the specificity is greater than 95%; most often, this will not be the case. This means that quite often conclusions about a particular psychiatric disorder that are based on an epidemiologic study will, in fact, be biased by the dilution of cases by at least one-third with subjects who are not disordered. This dilution renders the interpretation of risk factors, biological markers, and prognosis (to name a few) hazardous. Genetic marker and linkage studies are also affected by these considerations. In general, conclusions based on noncases in an epidemiologic study will be less biased because the predictive value negatives will be higher than the predictive value positives. The three hypothetical researchers would need to approach the choice of a cutoff differently.

Researcher A should consider the fact that a child who is falsely labeled as being in need of evaluation for depression (false positive) may be inconvenienced and unnecessarily worried, but a child with depression who is missed (a false negative) may be deprived of important therapy. Thus, the worry is more about false negatives in this type of a screening procedure. The cutoff should be set to maximize sensitivity without too much concern about the predictive value positive.

Researcher B's goal is to ascertain the same mix of patients that a clinician is likely to identify. False positives are of concern because one would not want to treat a child for a disorder that he or she does not have. However, false positives are likely to be identified as treatment progresses. False negatives are likely to be of somewhat more concern to a clinician, who would not want to leave a depressed child untreated. (Note that if this researcher were studying a new treatment that was not yet in clinical practice, she might want a much more

pure group in which to study its effects. Thus, she could increase the specificity and the predictive value positive at the expense of sensitivity. This would not accurately predict the functioning of the treatment if it were put into general clinical use, however.)

Researcher C's choice of a cutoff should reflect the desire to identify a group of children who suffer from recurrent, severe depressions. False negatives are not of major concern, but false positives might impair the ability to discern a familial pattern. The specificity and the predictive value positive are the crucial measures to be maximized.

Notice that the three examples cover a range of situations, from a situation in which one needs very stringent criteria (researcher C) that minimize false positives to a situation in which looser criteria (researcher A) that minimize false negatives are deemed optimal. Researcher B falls midway between these two. However, if researcher B were studying the use of a risky drug, she might want to operate with criteria similar to those used by researcher C; conversely, she might want to operate with criteria closer to those used by researcher A if she were studying a very safe treatment and the untreated disorder was felt to be quite severe.

DISCUSSION

Psychiatric research has benefited in the last two decades from careful attention to the establishment of standardized nosologies; this has been followed, at each step, by the development of instruments for categorizing patients according to these nosologies. The widespread use of structured interviews to categorize subjects according to the currently accepted nosologies (e.g., DSM-III-R and the ICD-9-CM) can be seen by opening any current psychiatric journal. For example, the March 1991 issue of the *American Journal of Psychiatry* had eight "Regular Articles" that involved categorizing subjects; six of these studies used a case definition procedure based on a structured interview. Similarly, the March 1991 issue of the *Journal of the American Academy of Child and Adolescent Psychiatry* had 22 "Articles," and 11 of the studies used a case definition strategy based on standard instruments. Our concern is that the results of structured interviews are being accepted as valid without explicit consideration of the consequences of choosing specific case definition strategies based on these instruments. Clearly, the utility of current psychiatric research depends on many methodologic considerations. The method of case definition is one that is often overlooked yet has broad implications. This holds true across many areas of research, including those which focus on epidemiology, pharmacology, and psychodynamic issues. It is worth considering, therefore, the types of case definition strategies that are in current use. Although these strategies are most often poorly defined in research reports, they can be seen to have many of the following features:

1. The choice of an external validator is either not mentioned or poorly justified.

2. The choice of a separator is frequently based on a "standard" instrument with published validity and reliability data that may or may not be related to the task at hand. When several instruments are used, the method of combination is either not explained or, if explained, not justified.

3. The choice of a cutoff is almost always based on the distribution of scores in a mixed population of disordered and nondisordered subjects, either a subset of the current subjects or subjects from a previous study. Frequently used criteria include a given upper or lower percentile or a certain number of standard deviations above or below the mean. Since the prevalence of the disorder in the study population and the separate distributions of results in the disordered and nondisordered groups are not reported, the rates of false positives and negatives cannot be determined. Similarly, the predictive value positive and negative cannot be ascertained. Thus, a reader of the report cannot determine the nature (or case mix) of the groups that are being described. When separators are combined, their function as a joint measure tends not to be described at all.

The lack of gold standards in psychiatry is frequently used as an explanation for the suboptimal methods being used (see, for instance, 1). It must be recognized, however, that the instruments being used are validated against some measure. The question is whether that measure is relevant to the current research. At the present time, explicit definition of the external validator being used would represent a marked improvement over current practice.

RECOMMENDATIONS

Future research must focus on better characterization of psychiatric disorders, which could lead to the development of better external validators and eventually perhaps to the elucidation of gold standards. In the meantime (and in order to allow this work to progress), the following steps are suggested.

1. Each step of the case definition process should be made explicit in research reports. Whenever possible, the basis for the decisions and their likely implications should be discussed.

2. Whenever possible, data should be reported in an uncategorized fashion (as well as categorized, if this is relevant to the work) so that information is not lost. This will allow those who wish to use the results of the research to choose a different case definition strategy to suit their own work. For example, a researcher may choose to use a certain cutoff for a diagnosis in one study, but the same data could be used by another researcher (or clinician) to choose a different cutoff for his or her application.

3. More attention should be paid to translating research data into clinical recommendations. This important step is frequently ignored; as a result, the case definition strategies used by researchers are implicitly accepted as relevant to clinical practice—even though it

is unlikely that many clinicians use these identical strategies in their practices.

This paper has focused on diagnostic decision making and the importance of explicitly considering the consequences of alternative strategies. Clinical decision making, in which the consequences of different possible interventions are considered, is equally important. Together they form the basis for clinical care. Decision analysis and clinical epidemiology provide a logical framework for this work.

The past 10 years have seen changes in psychiatry that make these endeavors particularly crucial. The range of therapies available for patients include more acute, more effective, and more dangerous interventions. The importance of diagnosis is therefore of increasing importance, but the consequences of false diagnosis or suboptimal therapeutic choices are consequently more extreme than in the past. In the policy arena, clinicians must defend their decisions more clearly and more often to administrators, who, in turn, must allocate limited resources to an ever-increasing array of treatment options. Residency training programs should be responsive to these changing demands on psychiatrists and should incorporate the basic principles of decision analysis and clinical epidemiology into their curricula.

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American Psychiatric Association Practice Guidelines

Practice Guideline for Eating Disorders

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PREFACE

STATEMENT OF INTENT

This guideline is not intended to be construed or to serve as a standard of medical care. Standards of medical care are determined on the basis of all clinical data available for an individual case and are subject to change as scientific knowledge and technology advance and patterns evolve. These parameters of practice should be considered guidelines only. Adherence to them will not ensure a successful outcome in every case, nor should they be construed as including all proper methods of care or excluding other acceptable methods of care aimed at the same results. The ultimate judgment regarding a particular clinical procedure or treatment course must be made by the physician in light of the clinical data presented by the patient and the diagnostic and treatment options available.

This practice guideline was published in February 1993.

REFERENCE CODING SYSTEM

The following coding system is used to indicate the nature of the supporting evidence in the summary recommendations and references:

- [A] Randomized controlled clinical trial, crossover design with randomly assigned treatment sequence
- [B] Nonrandomized case-control study, repeated measures design, follow-up study
- [C] Nonrandomized cohort study

- [D] Clinical report with nonrandomized historical comparison groups
- [E] Case report or series
- [F] Expert consensus
- [G] Data consolidation and reanalysis, e.g., meta-analysis
- [H] Epidemiologic report
- [I] Subject review
- [J] Other, e.g., published instrument, published abstract, published letter

LITERATURE REVIEW PROCESS

The following sources were reviewed:

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2. The following authoritative volumes published within the past decade:

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4. A MEDLINE computerized search was conducted in March 1990 using the following key words:
1. Eating Disorders and Treatment, yielding 151 references.
 2. Anorexia Nervosa and Treatment, yielding 188 references.
 3. Bulimia Nervosa and Treatment, yielding 115 references.
- Abstracts were reviewed for pertinent data-based studies.

Practice Guideline for Eating Disorders

American Psychiatric Association

I. DISEASE DEFINITION, EPIDEMIOLOGY, AND NATURAL HISTORY

These guidelines address anorexia nervosa and bulimia nervosa only; they do not address eating disorders not otherwise specified, pica, or rumination. The discussion of diagnoses in these guidelines are based on DSM-III-R criteria. Anorexia nervosa and bulimia nervosa affect large numbers of persons, with 90%–95% of cases occurring in females. With the obvious exception of concerns regarding menstrual function and female sexuality, issues of assessment and treatment for male patients (1) generally parallel those for females. DSM-III-R criteria for anorexia nervosa and bulimia nervosa are outlined below. Some aspects of diagnosis and treatment may require special consideration for the very young.

A. DSM-III-R CRITERIA

1. For anorexia nervosa

a. Refusal to maintain body weight over a minimal normal weight for age and height, e.g., weight loss leading to maintenance of body weight 15% below that expected; or failure to make expected weight gain during period of growth, leading to body weight 15% below that expected.

b. Intense fear of gaining weight or becoming fat, even though underweight.

c. Disturbance in the way in which one's body weight, size, or shape is experienced, e.g., the person claims to "feel fat" even when emaciated, believes that one area of the body is "too fat" even when obviously underweight.

d. In females, absence of at least three consecutive menstrual cycles when otherwise expected to occur (primary or secondary amenorrhea). (A woman is considered to have amenorrhea if her periods occur only following hormone, e.g., estrogen, administration).

2. For bulimia nervosa

a. Recurrent episodes of binge eating (rapid consumption of a large amount of food in a discrete period of time).

b. A feeling of lack of control over eating behavior during the eating binges.

c. The person regularly engages in either self-induced

vomiting, use of laxatives or diuretics, strict dieting or fasting, or vigorous exercise in order to prevent weight gain.

d. A minimum average of two binge eating episodes a week for at least 3 months.

e. Persistent overconcern with body shape and weight.

B. EPIDEMIOLOGY AND CHARACTERISTICS OF EATING DISORDERS

The prevalence of eating disorders appears to be increasing (2, 3) and may range from 1%–4% of adolescent and young adult women in predominantly white upper-middle- and middle-class student groups (3–7). Although the prevalence of these disorders elsewhere in the population is much lower (8), increasing numbers of cases are being seen in males, minorities, and women of all age groups. Some experts feel that increasing numbers of cases are being seen in prepubertal children. Homosexual men may be at greater risk than heterosexual men (9). Bulimia nervosa is more common than anorexia nervosa (10).

Weight preoccupation is a primary symptom in both anorexia nervosa and bulimia nervosa. Many patients demonstrate both anorexic and bulimic behaviors. Anorexia nervosa appears in restricting and bulimic subtypes; up to 50% of anorexia nervosa patients develop bulimic symptoms, significant numbers of patients who are initially bulimic develop anorexic symptoms, and restricting and bulimic subtypes may occasionally alternate in the same patient (11–16). For these reasons some consider the disorders to occur along a continuum. Patients with the restricting subtype ("dieters") limit energy intake to as few as several hundred kilocalories per day, limit food selection, and often demonstrate obsessive-compulsive symptoms regarding food and other matters. Patients with the bulimic subtype suffer from frequent eating binges, usually purge, and are often self-destructive (13). Patients with either subtype may exercise for hours daily (17) and may demonstrate bizarre food preferences, social isolation, diminished sexual interest, and depression. Anorexia nervosa patients who purge but who do not objectively binge eat are often encountered. Careful assessment of exactly what each patient means by a "binge" is imperative.

Physical complications of anorexia nervosa include all serious sequelae of malnutrition, including cardiovascular compromise. Prepubertal patients may have arrested sexual maturation, general physical development, and growth and may not grow to anticipated heights. Even patients who look and feel deceptively well and who have normal ECGs often have bradycardia and other manifestations of impaired cardiac function, such as drop in orthostatic blood pressure and increase in pulse rate, and may be prone to sudden death (18). Prolonged amenorrhea (more than 6 months) is associated with potentially irreversible osteopenia and a correspondingly higher rate of pathological fractures (19). Patients may suffer from dehydration, electrolyte disturbances, gastrointestinal motility disturbances, infertility (20), hypothermia and other evidence of hypometabolism, and from the psychological sequelae of starvation described later in the text.

Although laboratory findings may be normal in spite of profound malnutrition, abnormalities may include neutropenia with relative lymphocytosis, abnormal liver function, hypoglycemia, hypercortisolemia, hypercholesterolemia, hypercarotenemia, low serum zinc levels, widespread disturbances in endocrine functioning (including low T_3 levels which are reversible with weight restoration and generally should not be treated with replacement therapy), and electrolyte disturbances (21–23). Abnormal computerized tomography (CT) scans of the brain may be found in more than half of patients with anorexia nervosa (24), and patients with weight loss may exhibit decreased metabolic rate (25).

Physical complications of bulimic behaviors include electrolyte disturbances (notably, a hypokalemic, hypochloremic alkalosis in patients who vomit), mineral and fluid imbalances, hypomagnesemia, gastric and esophageal irritation and bleeding, large bowel abnormalities due to laxative abuse, erosion of dental enamel, parotid enlargement, and accompanying hyperamylasemia. Mallory-Weiss esophageal tears occur rarely. Abuse of ipecac to induce vomiting may cause cardiomyopathies (with sudden death) or peripheral muscle weakness (26). Resting bradycardia, hypotension, and decreased metabolic rate are observed in some bulimic patients and may reflect decreased activity in the sympathetic nervous system and the thyroid axis (27). In addition, although bulimic patients may appear physically within the standards of healthy weight, they may show psychological correlates of starvation, so that definitive psychological assessment may be difficult to accomplish before eating and weight are stabilized.

Symptoms of eating disorders are seen in heterogeneous psychiatric populations suffering from varied types and degrees of psychopathology, character organizations, and levels of ego functioning (15, 16, 28–32). Early histories of patients with eating disorders are often complicated by medical and surgical illnesses, separations, family deaths, and behavioral disturbances. Whether the prevalence of these problems is higher among persons with eating disorders in comparison to those with other forms of psychopathology is not known. Sexual abuse has been reported in 20%–50% of patients with bulimia nervosa (33), but this rate may be similar to that found in other psychiatric populations (34). For those who have been victimized, the abuse is a major treatment consideration and assessment for abuse is very important (35).

Patients with relatively uncomplicated eating disorders are encountered in college populations and among younger age groups, but many patients seeking treatment at tertiary psychiatric treatment centers are far more complex. Comorbid major depression and/or dysthymia have been reported in 50%–75% of anorexia nervosa patients (28). In addition, obsessive-compulsive disorder may be found in about 10%–13% of cases (28, 36), with a lifetime prevalence of obsessive-compulsive disorder in anorexia nervosa of about 25% (28). Among patients with bulimia nervosa, increased rates have been reported for anxiety (43%) and chemical dependency (49%) disorders (37), bipolar disorder (12%) (38), and personality disorders (or at least substantial personality trait disturbances) (50%–75%) (39–41). Due to disputes regarding diagnostic criteria, disagreement exists regarding comorbidity rates for borderline personality disorder and eating disorders, with reported estimates varying widely between 2% and 60% (42). Additionally, many bulimic eating-disordered patients have dissociative symptoms, sexual conflicts and disturbances, and a variety of impulsive behaviors that frequently involve overspending, shoplifting, promiscuity, and self-mutilation (15, 16, 32, 43).

First-degree female relatives of patients with anorexia nervosa have increased rates of anorexia nervosa (44). Twins of patients with bulimia nervosa also have increased rates of bulimia nervosa, with monozygotic twins having higher concordance than dizygotic twins. The evidence regarding rates of bulimia in other first-degree female relatives is controversial (3). In addition, families of patients with bulimia nervosa have increased rates of substance abuse (particularly alcoholism) (45), affective disorders (37), and obesity (46).

II. TREATMENT PRINCIPLES AND ALTERNATIVES

A. GOALS OF TREATMENT

Treatment interventions are first aimed at nutritional rehabilitation and the restoration of normal eating patterns to correct the biological and psychological sequelae of malnutrition that may perpetuate eating-disordered behavior. The concurrent longer-term goals are to diagnose and help resolve the associated psychological, family, social, and behavioral problems so that relapse does not occur.

1. *Malnutrition and other biologically mediated problems*

Consensus currently exists that many of the physical and psychological symptoms of eating disorders may result from malnutrition (47). Volunteers who submit to starvation and semistarved prisoners of war develop food preoccupation, food hoarding, abnormal taste preferences, binge eating and other disturbances of appetite regulation, symptoms of depression, obsessiveness, apathy, irritability, and other personality changes. These disturbances reverse with refeeding, although it may take considerable time following weight restoration for them to abate completely (48). Complete psychological assessment may not be possible until some degree of weight normalization is achieved (49).

Although biological hypotheses have suggested that primary hypothalamic or suprahypothalamic abnormalities account for profound disturbances in hormones, neurotransmitters, and neuromodulators (50–53), and other hypotheses have suggested that eating disorders may be variants of affective disorders (54, 55), all of these hypotheses are rendered somewhat questionable because virtually all of the biological disturbances and many of the mood disturbances abate with nutritional rehabilitation (44, 56, 57). However, treatment studies suggest that, at least for some patients, specific relationships may exist between mood, bulimic behaviors, and responses to specific antidepressant medications (58).

2. *Psychological, behavioral, and social deficits*

Formulations regarding psychological abnormalities seen in patients with eating disorders are based on psychodynamic (30, 59, 60), psychoanalytic (15, 32, 61, 62), cognitive (63, 64), learning (65), family systems (66–68), sociocultural (69, 70), and feminist (71) theories. One prominently held view asserts that central psychological features of patients with eating disorders include a sense of pervasive ineffectiveness which results in an attempt to gain self-control in the sphere of weight, difficulties in interpreting inner sensations, including hunger and satiety, and difficulties in both in-

terpreting and tolerating many affective states. Deficits in self-structure, self-esteem, self-coherence and self-regulation, and incomplete and ambivalent object relations may leave these patients ill-equipped for the developmental tasks of separation/individuation and result in a weak sense of personal and gender identity and in a pervasive sense of ineffectiveness and helplessness. The relative contributions of autonomous constitutional factors, developmental difficulties with separation/individuation, difficulties with self-esteem regulation, pathogenic family styles of interaction, and pathogenic social input are thought to be important but have not been empirically established (72). Preoccupation with appearance and weight may become the focus for attempts at mastery during developmentally stressful periods such as adolescence. Women with greater degrees of conflict regarding maturation, separation, sexuality, self-esteem, or compulsivity or greater difficulties in tension regulation may be more prone than others to develop eating disorders. For example, anorexia nervosa can provide a means for avoiding both physical and psychological aspects of sexuality (30, 59). Initially, patients may be rewarded for thinness by family and peers, and peers may even compete in this regard. However, as symptoms become habitual, many patients experience a growing sense of the eating disorder becoming their core identity. Other patients persistently deny the abnormality or the severity of their eating disorders.

3. *Culturally mediated distortions*

Constructs such as self-worth and attractiveness have become closely associated with dieting and weight control for women in Western culture (69, 70, 73) and among immigrant women undergoing rapid cultural change (74). Challenging distorted values related to shape without attacking individual bases for self-esteem is a delicate task requiring clinical sensitivity (64, 75).

B. TREATMENT OF ANOREXIA NERVOSA: INDICATIONS, EFFICACY, AND SAFETY

Anorexia nervosa is a medically, psychopathologically, and interpersonally complex, serious, and often chronic condition that requires ongoing commitment and attention to the multiple, interdigitating diagnoses and to a comprehensive treatment plan that involves medical management, individual psychotherapy, and family therapy.

At the present time the best initial results appear linked to weight restoration accompanied by individual and family psychotherapies when the patient is medically ready to participate.

1. Nutritional rehabilitation and treatment setting

There is general agreement that weight restoration should be a central, early treatment goal for the seriously underweight patient (76). Weight restoration per se in these patients may result in improvement in obsessional thinking, mood, and personality disturbance.

a. Target weights. The ultimate weight target should be a return to an individually determined healthy body weight, one at which normal reproductive function resumes (77–79) and bone demineralization is reversed (80). The relationship between an individual's healthy weight and "ideal weights" published in standard tables (e.g., Metropolitan Life Insurance Company 1983 [81], National Center for Health Statistics 1973 [82]) is quite variable; some patients have always been slim and others may require a weight of 115% or more of published ideal weight for height to achieve a healthy status. Weight at discharge in relation to the healthy target weight may vary depending on the patient's ability to feed herself, her motivation and ability to participate in aftercare programs, and the adequacy of aftercare, including partial hospitalization.

Because several different "standards" of ideal body weight exist and healthy weights may be more properly understood in terms of ranges rather than specific numbers, many eating disorders consultants are moving toward the use of the body mass index as a standard measure of nutritional status rather than percentages of ideal body weight: $\text{body mass index} = \text{weight (kg)} / \text{height (m)}^2$. Healthy ranges for body mass index are related to the age of the patient, and appropriate tables should be consulted (83, 84).

b. Choice of setting. Although some underweight patients who are less than 20% below average weight for height may be successfully treated outside of the hospital, such treatment usually requires a highly motivated patient, cooperative family, and brief duration of symptoms. Such patients may be treated in outpatient programs with close monitoring for several weeks to assess their response (85–87). Most severely underweight patients and those with physiological instability require inpatient medical management and comprehensive treatment for support of weight gain. Decisions to hospitalize on a psychiatric versus general medical or adolescent/pediatric unit depend on the patient's medical status and on the skills and abilities of local psychiatric and medical staff and local programs to care for the patient's medical and psychiatric problems (88).

Increasingly, partial hospitalization day hospital programs are being utilized in attempts to decrease the length of some inpatient hospitalizations and, for milder cases, in place of hospitalization. However, such programs cannot always easily replace traditional hospital programs or shorten lengths of stay, especially for patients with lower initial weights (e.g., those who are 70% of average weight for height, or below) (89). In order to benefit from partial hospitalization, patients must be motivated to participate in an intensive treatment program with mutually agreed-upon expectations

of symptomatic change, including weight gain and/or decreased binge eating or purging, and must also demonstrate ability to relate in a group setting (90).

c. Inpatient programs. Regardless of the treatment setting, the availability of staff trained in and knowledgeable about the care of persons with eating disorders is critical. Where the staff does not have the training or experience to deal with patients with eating disorders, the psychiatrist or other qualified professionals must spend time educating, consulting with, and supervising the staff and managing their reactions to the patient's condition. Such work with the staff, though time consuming, is essential to the success of the treatment (91).

Both positive (e.g., praise) and negative (e.g., restriction of exercise or bed rest) reinforcers can influence the rates at which patients eat and gain weight, and combinations of informational feedback regarding weight gain and caloric intake, large meals, and behavioral programs may produce good short-term therapeutic effects (92). Moreover, a meta-analysis of treatment programs using medications or psychotherapy suggested that medication programs alone have failed to produce consistent weight gain in anorexia nervosa. Programs utilizing behavior therapy were more efficient, resulting in shorter hospital stays (92). However, the speed of weight gain during inpatient treatment is no assurance of long-term outcome.

Some studies have shown that "lenient" behavioral programs utilizing initial bed rest and the threat of returning the patient to bed if weight gain does not continue may be as effective and perhaps in some situations more efficient than "strict" programs in which meal-by-meal caloric intake or daily weight is tied precisely to a schedule of privileges such as time out of bed, time off the unit, and permission to exercise or receive visitors (93, 94). Lenient programs are more likely to enlist the patient's cooperation and sense of participation and control; they also increase staff satisfaction by reducing the policing functions. In strict programs, staff are more likely to develop conflicted relationships with patients that interfere with supportive and empathic aspects of the treatment process. The relative merits of various degrees of strictness and leniency require further study. However, some basic milieu practices may be necessary, such as routinely restricting unaccompanied use of the bathroom for a period of time following meals to discourage purging. These practices may be individualized depending on the patient's past history and personality structure.

Most consultants believe that nasogastric tube feeding or even total parenteral nutrition may be required only rarely and in life-threatening situations. There is significant recognition of the danger of rapid refeeding (e.g., severe fluid retention and cardiac failure) and of forced nasogastric or parenteral feeding. These interventions should not be used routinely. However, some severely malnourished anorexic patients may accept nasogastric feeding more willingly than eating, especially in the early stages of renourishment. In situations where forced feeding is considered, careful thought

should be given to clinical circumstances, family opinion, and relevant legal and ethical dimensions of the patient's treatment.

d. Hospital stay. Research addressing optimal length of hospitalization is sparse. In one study, significantly fewer relapses were observed in patients able to complete an inpatient treatment program (discharged at normal weight) compared with those who left the hospital before completing treatment (95).

2. Psychosocial treatment

The exact role of psychotherapy in the acute treatment of the hospitalized, severely malnourished patient remains unclear. Although some studies question the utility of individual or family psychotherapy during the acute refeeding stage (96), there is general consensus that the patient and her family have to be engaged from the very beginning of treatment and should be educated as to the nature of the patient's condition, the relationships among semistarvation and the symptoms of anorexia nervosa, psychodynamic, family, and sociocultural issues, and related matters (97, 98). This engagement can serve as a foundation for the later use of more insight-oriented therapies. Family or individual/couples psychotherapy for parents is frequently useful in helping young patients achieve age-appropriate separation and symptom alleviation. One study showed that 1 year following discharge from the hospital, patients with anorexia nervosa with onset at or before age 18 and with a duration of fewer than 3 years showed greater improvement with family therapy than individual psychotherapy; in contrast, older anorexia nervosa patients did better with individual therapy than with family therapy (99). However, patients in this study were not assigned to *both* family and individual treatment, a combination frequently used in practice.

Since many patients have difficulty talking about their problems, clinicians have also tried a variety of nonverbal therapies, such as creative arts therapies, and have reported them to be useful (100).

Consultants agree that having the same psychiatrist treat the patient throughout hospitalization and aftercare, using therapeutic modalities that best fit the circumstances, constitutes a desirable approach whenever practical.

Many types of therapies have been reported to be of value in case series (49). Although intensive psychodynamic psychotherapy may sometimes be ineffective with emaciated patients during the acute weight restoration phase, many consultants see psychodynamic or interpersonal psychotherapies as very useful for subsequent psychological maturation during weight maintenance. The experience of a large number of psychoanalytically trained psychiatrists suggests that good results may be obtained by psychoanalysis or psychoanalytic psychotherapy in nondebilitated patients when underlying personality disorders are important in contributing to the illness. Psychiatrists treating patients from a strictly psychoanalytic perspective will focus on

longer-term treatment-oriented goals, not focused on, but resulting in, weight restoration. The experience of these clinicians suggests that in most cases their results are as rapid as, and more durable than, most other methods of treatment (32, 101). Many clinicians favor cognitive-behavioral psychotherapies for maintaining healthy eating behavior and cognitive or interpersonal psychotherapies for inducing mature insights and promoting more effective coping (102, 103). The use of various modalities considered coercive by patients with anorexia nervosa, for whom control is of such importance, is an issue to be carefully weighed.

3. Medications

Few controlled studies of the use of medications for anorexia nervosa have been published. In one study lower-weight patients with the restricting subtype who were receiving intensive inpatient treatment seemed to benefit, albeit to a small degree, from cyproheptadine; amitriptyline had some value as well (104). Results from studies with lithium (105), clomipramine (in lower-than-usual doses) (106), and pimozide (107) have been unimpressive. Because of reported increased seizure risk associated with bupropion in patients with eating disorders, this medication cannot be recommended for such patients (108, 109). There have been no controlled pharmacologic studies conducted solely with child or early adolescent populations of patients with eating disorders.

Many different somatic treatments ranging from vitamin and hormone treatments to electroconvulsive therapy have been tried in uncontrolled studies. None has been shown to have specific value (23). Medications used most often on an empirical basis include antidepressants for patients with depressions that persist in spite of or in the absence of weight gain; low doses of neuroleptics for marked obsessiveness, anxiety, and psychotic-like thinking; and antianxiety agents used selectively before meals to reduce anticipatory anxiety concerning eating (110, 111). Uncontrolled trials have suggested that fluoxetine may help some patients in weight restoration (112) and weight maintenance phases (113), but many patients do not improve with this or any other currently available medication. Although fluoxetine has been reported at higher doses (e.g., 60 mg/day or more) to impair appetite and cause weight loss in normal-weight and obese patients, this effect has not been reported in anorexia nervosa patients treated at lower doses.

Most consultants 1) find that malnourished depressed patients are more prone to the side effects of and less responsive to antidepressant medications than other patients with depression; 2) are concerned that tricyclic antidepressants may add to the risk of hypotension and arrhythmia in anorexia nervosa patients, particularly in purging anorexia nervosa patients whose hydration may be inadequate and whose cardiac status may be nutritionally compromised; and 3) find that at least some symptoms of depression remit with weight

gain. However, for patients with persistent depression the use of antidepressants should always be considered; these medications may be helpful if not contraindicated by cardiovascular status. For patients in whom potential cardiovascular effects of medication are of concern, consultations to evaluate cardiovascular status and to advise on the use of medication may be helpful.

Estrogen replacement to reduce calcium loss and thereby reduce the risks of osteoporosis is sometimes used in anorexia nervosa patients with chronic amenorrhea and should be considered (114). However, for adolescent patients, authorities advocate waiting at least 1 year before offering estrogen replacement (115), during which time efforts should be made to increase weight and achieve resumption of normal menses.

4. Application of the addiction model

Given the high prevalence of substance abuse among persons with eating disorders and the likelihood that either condition may precipitate the other, where substance abuse exists it is important that a progressive treatment plan be initiated for this disorder as well (116).

Some clinicians consider that eating disorders may be usefully treated via addiction models, but no data from short- or long-term outcome studies with these methods have been reported. These programs should be equipped to care for patients with substantial psychiatric and/or general medical problems associated with their eating disorders.

C. PROGNOSIS IN ANOREXIA NERVOSA

Reviews of carefully done follow-up studies conducted on hospitalized or tertiary referral populations at least 4 years after onset of illness show that about 44% of patients had an overall good outcome (weight restored to within 15% of recommended weight for height and regular menstruation established), about 24% had poor outcome (weight never approached 15% under recommended weight for height and menstruation was absent or at best sporadic), about 28% had an intermediate outcome (between that of the good and poor groups), and fewer than 5% had died (early mortality). Poorer prognosis has been associated with initial lower minimum weight, the presence of vomiting, failure to respond to previous treatment, premorbidly disturbed family relationships, and marital status (being married) (117, 118). Mortality, primarily resulting from cardiac arrest or suicide, increased with length of follow-up and reached about 20% among patients followed for more than 20 years (119). Furthermore, about two-thirds of patients continued to have persistent morbid food and weight preoccupations, up to 40% had bulimic symptoms, and many had dysthymia, social phobia, obsessive-compulsive symptoms, and/or substance abuse (28). Patients with less severe degrees of illness, whose conditions therefore permit them to be treated primarily as outpatients, tend to have better

outcomes. However, the chronic nature of anorexia nervosa often requires that even patients with less severe illness participate in long-term maintenance treatment programs to prevent relapse.

D. TREATMENT OF BULIMIA NERVOSA: INDICATIONS, EFFICACY, AND SAFETY

Strategies for the treatment of bulimia nervosa include nutritional counseling and rehabilitation; individual and/or group cognitive-behavioral, behavioral, psychoanalytic, and psychodynamic approaches; family interventions; and medications (120).

1. Hospitalization

Most consultants currently hold that hospital treatment for uncomplicated bulimia nervosa is rarely necessary and that such patients should first be treated in outpatient or day hospital programs. Hospitalization should be considered in cases complicated by suicidality, severe concurrent alcohol or drug abuse, or life-endangering medical problems not amenable to outpatient treatment. For those whose eating behavior is entirely out of control and who do not make substantial progress during an adequate trial of outpatient treatment, hospital admission aimed at breaking the binge-purge cycle may prove helpful. These periods of stabilization often require several weeks.

2. Psychosocial treatment

Patients treated for bulimia nervosa appear to improve; however, the number of patients who achieve full abstinence from binge-purge behavior is highly variable, with the minority becoming fully abstinent, according to most published studies. Many therapeutic approaches including individual cognitive-behavioral therapy, behavior therapy, focal psychotherapy (121), psychodynamic and interpersonal psychotherapy, psychoanalysis, and addiction-oriented therapy have value (122–128). Nevertheless, current research emphasizes cognitive-behavioral therapy for symptom reduction, at least in the short run (129). One study found that although cognitive-behavioral therapy, interpersonal psychotherapy, and behavior therapy were all effective in reducing binge eating and depressive symptoms, cognitive-behavioral therapy was more effective than the other two in modifying outpatients' disturbed attitudes toward shape, weight, dieting, and the use of vomiting to control shape and weight (129). When cognitive therapy was compared to short-term focal psychotherapies both were effective, but patients treated with cognitive-behavioral therapy had greater overall improvement (121). Clinicians unfamiliar with this approach may benefit from cognitive-behavioral therapy treatment manuals for bulimia nervosa (130–134). Still, given the complexities of concurrent psychopathologies in patients with bulimia nervosa, many therapeutic strategies have a role, and individual psychodynamic

and interpersonal, family-oriented, and psychoanalytic therapies, which many clinicians find useful and which have yet to be used as comparison treatments in any well-designed studies, may be particularly useful for long-term functioning. For example, as eating disorders symptoms abate, some patients with prior histories of traumatization and abuse may experience an intensification of other symptoms, including posttraumatic stress disorder symptoms (135).

Conflicting evidence exists as to whether the behavioral procedure of exposure (binge eating food) plus response prevention (inhibiting vomiting) (136, 137) is superior to, adds to, or subtracts from the efficacy of cognitive-behavioral therapy alone, and evidence is available to support each of these viewpoints (138–140).

Many controlled group psychotherapy studies of bulimia have been reported, all showing treatment superior to no treatment (141–147). A recent meta-analysis of 40 group treatment studies of bulimia suggested moderate efficacy, with improvement typically maintained in those studies reporting 1-year follow-up data (148). Larger posttreatment effect sizes are associated with more hours of therapy per week and with the addition of other treatment components such as individual therapy (149). Although some reports suggest that group treatments tend to have a higher dropout rate compared to individual treatments (150), this finding has been questioned. There is some evidence that treatments that include dietary counseling and management as part of the program perform better than those that do not (125, 151), and available evidence suggests that more frequent visits early in treatment produce better results (63, 129, 152).

Virtually every type of individual psychotherapy for the treatment of bulimia nervosa has been described in uncontrolled case series, and many seem to help (49). In practice, many consultants use psychodynamically oriented and interpersonal psychotherapies after initial symptom control and, at times, to help patients with initial symptom resolution. In properly selected cases, psychoanalytic psychotherapy and psychoanalysis have been reported to successfully address the psychological conflicts associated with patients' symptoms and to lead to symptom alleviation (32). Family therapy was reported as helpful in a large case series (153). Although no systematic studies exist, some patients have found Overeaters Anonymous and similar groups to be helpful in recovery, in part because of the networking, sense of connectedness to a group, and 24-hour-per-day support against food cravings that they offer (154, 155). At the same time, controversy exists regarding the role of 12-step programs that do not address nutritional considerations and psychological/behavioral deficits when used as the sole intervention in the treatment of eating disorders (128).

3. Medications

Double-blind placebo-controlled studies have demonstrated the efficacy of imipramine (156, 157), desipramine

(158), trazodone (159), and fluoxetine (160, 161) in reducing bulimic symptoms. In the imipramine studies, the large majority of patients reduced their eating binges by at least half, and about a third became free of binge eating and purging. In the desipramine study about two-thirds of those who achieved therapeutic blood levels experienced a remission in bulimic symptoms. This is the only study thus far to demonstrate a relationship between serum drug level and symptom response in bulimia. Furthermore, the desipramine study involved only nondepressed bulimic patients.

The monoamine oxidase inhibitors (MAOIs) phenelzine (162) and isocarboxazid (163) have been shown to effectively reduce bulimic symptoms. Recent data suggest that patients with atypical depression and bulimia may preferentially respond to phenelzine in comparison to imipramine (58). Because some patients have difficulty avoiding foods containing tyramine, a careful evaluation of the patient's reliability in maintaining a tyramine-free diet is indicated when MAOIs are being considered (111, 164). Carbamazepine (165) and lithium have been less effective in treating the symptoms of bulimia (166, 167), and their occasional adjunctive use in patients with eating disorders should rest on consideration of other comorbid conditions.

To summarize, antidepressant medications can be useful in the treatment of bulimia nervosa. Doses of tricyclic and MAOI antidepressants used to treat bulimia nervosa are generally at the same levels as those used to treat mood disorders. However, higher doses of fluoxetine (60 mg/day) seem more effective than lower doses (20 mg/day) (160). Several medication trials are sometimes required to establish the proper medication for a given patient (168, 169).

4. Psychosocial and/or medication strategies

Few studies have compared psychosocial and medication treatments (170, 171). In one study in which patients were randomly assigned to treatment or placebo groups, intensive group outpatient treatment, imipramine, and imipramine plus intensive group outpatient treatment were all better than placebo, and intensive group outpatient treatment was superior to imipramine alone in reducing binge eating, purging, and symptoms of depression (170). The intensive group outpatient treatment included many hours per week of structured meetings and lectures, and meals were provided for several weeks. The program was based on cognitive-behavioral and educational principles. Adding imipramine to intensive group outpatient treatment did not improve outcome with respect to eating behaviors per se, but did improve outcome for symptoms of depression and anxiety. However, use of active drug was also associated with a higher dropout rate.

In a study in which patients were randomly assigned to cognitive-behavioral therapy alone, cognitive-behavioral therapy plus desipramine, or desipramine alone, both cognitive-behavioral therapy groups were superior to desipramine alone. At 24 weeks, cognitive-be-

havioral therapy in combination with desipramine (but not cognitive-behavioral therapy alone) was superior to 16 weeks of desipramine alone in reducing binge eating and purging, dietary preoccupation, and hunger (171).

However, the use of a single antidepressant medication in these comparisons does not reflect actual clinical practice. Typically, when one antidepressant fails, a clinician will try a second or third agent, and this full range of choices often results in better antidepressant efficacy than was found in the studies mentioned (159, 168). Other medications, including narcotic antagonists and tryptophan, have been used but have received less systematic study; these medications should rarely be necessary.

E. PROGNOSIS IN BULIMIA NERVOSA

Little is known about the natural history or long-term outcome of bulimia nervosa. The overall short-term success rate for patients receiving psychosocial or medication treatment varies; patients have been reported to have a 50%–90% reduction in binge eating and purging, with an average of about 70% of those who com-

plete the treatment programs reporting substantial reduction of bulimic symptoms. Those treated as outpatients seem to maintain symptomatic improvement over follow-up periods of up to 6 years; however, some symptoms often persist (172, 173). Patients who function well and have milder symptoms at the start of treatment, and so are more likely to be treated as outpatients, often have a better prognosis than those who function poorly and have disabling symptoms. In contrast, at 3 years about 27% of patients hospitalized with bulimia have a good outcome (binge eating and purging less than once a month), 40% have an intermediate outcome, and 33% have a poor outcome (daily binge eating and vomiting or ongoing cathartic-diuretic abuse) (174). It is also well known that anorexia nervosa patients who purge are at much greater risk for developing serious medical complications (175). Very little is known about the prognosis of untreated bulimia nervosa. Over a 1- to 2-year period, bulimic patients who were never treated have reported modest degrees of spontaneous improvement, with roughly 25%–30% reductions in their overall levels of binge eating, purging, and laxative abuse (176, 177).

III. RECOMMENDATIONS

The following recommendations are based upon the degree and quality of the research data and/or clinical consensus.

A. GENERAL PRINCIPLES OF ASSESSMENT OF EATING DISORDERS

The following are recommended:

1. *Comprehensive multidimensional assessment.* At the very outset, clinicians should attempt to build trust, establish mutual respect, and develop a therapeutic relationship with the patient that will serve as the basis for ongoing exploration and treatment of the problems associated with eating disorders. During data gathering, clinicians and patients may initially be helped by semistructured interview instruments, such as the Eating Disorders Examination (178), or by the many well-regarded self-report questionnaires, such as the Eating Disorders Questionnaire (179), the Diagnostic Survey for Eating Disorders (180), the Stanford Eating Disorders Questionnaire (181), or the Eating Disorders Inventory (182), although their specific validity for populations of children and young adolescents has not been reported [E,F]. The complete assessment usually requires at least several hours, and often patients and their families may not initially reveal pertinent information about sensitive issues even when directly questioned. Some important information may be uncovered

only during ongoing treatment, after a trusting relationship has been established and the patient is better able to accurately identify inner emotional states.

a. *Eating disorder signs and symptoms.* Initial assessment generally includes a longitudinal history regarding lifetime actual and desired weights in relation to height; onset and patterns of menstruation; food restriction and avoidances; frequency and extent of binge eating, self-induced vomiting, and spontaneous vomiting; use of laxatives, diuretics, diet pills, and ipecac; and body-image and self-image disturbances. Food intake, food preferences and peculiarities, attitudes toward food, cognitive distortions regarding food and appearance, ritualistic and compulsive behaviors regarding food and exercise, and details of other associated behavioral, psychological, and social impairments should generally also be assessed. It is often helpful in understanding the patient's problems to explore the patient's understanding of how the illness developed and the effect of any interpersonal relationships on the onset of the eating disorder. For patients whose recollections are vague, taking the detailed history of a single day or using a calendar as a timeline prompt may help elicit specific information. Much useful information is often obtainable by a less structured, open-ended approach. Family

history should be obtained regarding eating disorders and other psychiatric disorders, obesity, family interactions in relation to the patient's disorder, and attitudes toward eating, exercise, and appearance [E,F]. It may be helpful to involve health professionals who routinely work with children, parents, and school personnel in the assessment of young patients.

b. Psychiatric history. Attention should be paid to concurrent psychiatric disturbances, especially affective and anxiety disorders, suicidality, substance abuse, obsessive and compulsive symptoms, and personality disturbances. Shoplifting, stealing food, and self-mutilatory behaviors should be noted. A developmental history should attend to temperament, sexual and physical abuse, and sexual history. Psychological testing may clarify personality/neuropsychological disturbances. In addition to assessing behavioral and formal psychopathological aspects of the case, it is generally useful to investigate psychodynamic and interpersonal conflicts that may be relevant to understanding and treating the patient's eating disorder and to assess the patient's potential (e.g., motivation, self-awareness, availability of affects) for psychodynamic and interpersonal psychotherapies [E,F].

c. Physical health status. A full physical examination should be performed with particular attention to vital signs, weight for height, skin, the cardiovascular system, and evidence of laxative or diuretic abuse and vomiting. A dental examination should be performed. It is generally useful to assess growth, sexual development, and general physical development in younger patients. Laboratory studies should be determined on an individual basis depending on the patient's condition and as necessary for making treatment decisions. For ambulatory patients who are not obviously underweight, tests may include a complete blood count, urinalysis, BUN/creatinine levels, and electrolyte balance. For malnourished and severely symptomatic patients, laboratory studies often also include measures of calcium, magnesium, phosphorus, amylase, and liver function and an ECG. In all cases, if the patient is more than 15% below healthy body weight or if weight loss has been rapid, significant physical compromise may be present even if the ECG, laboratory studies, and physical examination appear normal [E,F].

d. Family assessment. Assessment of the family is important whenever possible for patients of any age living at home and for others who are so enmeshed with their families as to preclude all efforts to function independently. Family assessment may be extremely useful for some patients in order to understand interactions that may contribute to ongoing illness or that may potentially facilitate recovery [E,F].

2. Coordinated care plan. The coordinated care plan requires the collaboration of a variety of professionals to provide nutritional counseling and dental assessment, work with the family, and set up behavioral programs. Other physician specialists and dentists should be consulted when necessary for management of medical (e.g., cardiac dysfunction) and dental complications [E,F].

Other options which may be recommended in individual circumstances:

1. Imaging techniques and evaluation of brain function. Although abnormalities are frequently found on magnetic resonance imaging (MRI) and CT scans of the head and EEG, the therapeutic implications of these findings are unclear. Consequently, these tests are not routinely indicated. Consultants are of mixed opinions as to whether abnormalities on such tests are likely to increase patient compliance with treatment; the large majority believe that they have little value in this regard, but others believe that presenting patients with objective evidence of brain abnormalities may increase compliance with treatment [F].

2. Other laboratory tests. Depending on potential treatment decisions that may be heavily influenced by laboratory findings, the following tests may sometimes be of value: serum amylase levels as a possible indicator of persistent or recurrent vomiting behavior; estradiol levels in amenorrheic patients (low values are suggestive of bone loss); bone mineral densitometry to assess risk for pathological fractures secondary to osteoporosis in chronic anorexia nervosa; and levels of luteinizing hormone (LH) and follicle-stimulating hormone (FSH) when amenorrhea persists at normal weight [E,F].

3. Modification of approaches. Circumstances that may require modification of the assessment and treatment approaches described in these guidelines include problems in the availability of resources, treatment-reluctant or treatment-resistant patients, substantial problems of comorbidity, and particularly difficult family/social problems [F].

B. TREATMENT OF ANOREXIA NERVOSA

The following are recommended:

1. Initial treatment setting. A trial of outpatient treatment or partial hospitalization is warranted for highly motivated patients who have good social supports, are not losing weight rapidly, are metabolically stable, whose weight is not below 70% of average weight for height, and when close monitoring of physical state can be guaranteed [E,F]. However, many patients require hospital treatment. Patients with rapidly falling weight or metabolic instability need to be hospitalized earlier in the course of care than others, as may children and young adolescents [E,F]. Legal interventions may be necessary to ensure the safety of treatment-reluctant patients whose medical conditions are life-threatening.

2. Aims of treatment. The aims of treatment should be to 1) restore patients to a healthy weight (at which menses will generally resume); 2) restore healthy eating patterns; 3) treat/remediate physical complications; 4) address dysfunctional thoughts, feelings, and beliefs; 5) correct defects in affect and behavioral regulation; 6) improve associated psychological difficulties; 7) enlist family

support of treatment where appropriate; and 8) prevent relapse. Many consultants believe that patients are less likely to relapse if they are hospitalized until they achieve healthy weight [E,F]. Those patients who are fully cooperative with their treatment, are achieving treatment goals, and for whom good aftercare is available may be discharged before full healthy weight is restored, with the plan that additional weight will be gained during aftercare [E].

Hospital programs should establish healthy target weights and expected rates of controlled weight gain to reassure patients that they can develop control over their own eating patterns. A supportive, encouraging staff with whom patients and families may develop realistic, trusting relationships is essential. Expectations for a reasonable rate of weight gain (e.g., 1–3 lb/week on inpatient units) and some positive and negative reinforcements (e.g., required bed rest, restriction of off-unit privileges, exercise contingent upon weight gain) should be built into the program [C,D,E,F,G]. Medical monitoring during refeeding should include vital signs, food and fluid intake and output, and observation for edema, rapid weight gain associated primarily with fluid overload, congestive heart failure, and gastrointestinal symptoms [E,F]. Outpatient programs often establish expectations of weight gain in the range of 1/2–2 lb/week [F]. A patient suspected of artificially increasing her weight should be weighed in the morning after voiding, wearing only a gown; her fluid intake should be carefully monitored. Physical activity should be adapted to the food intake and energy expenditure of the patient, taking into account bone mineral density and cardiac function. The focus of an exercise program should be on physical fitness as opposed to expending calories. Staff should help patients deal with their concerns about weight gain and body image changes, as these are particularly difficult adjustments for patients to make [F].

3. *Meal selection and caloric intake.* Although it is most desirable to help patients to eventually choose their own meals and to not avoid any of the major food groups, initially meal selection may be best recommended by a dietitian. Usual starting intakes of 30–40 kcal/kg per day (approximately 1000–1600 kcal/day) may ultimately have to be increased to as high as 70–100 kcal/kg per day for some patients during the weight gain phase, with 40–60 kcal/kg per day during weight maintenance [C,E]. Some patients who require higher caloric intakes are exercising frequently, vomiting, or discarding food, while others may have a truly elevated metabolic rate. Nutritional assessment, education, and ongoing support are essential [A,B].

4. *Medications.* Medications should not be used routinely [A,C,D,E,F]. The role for antidepressants is usually best assessed following weight gain, when the psychological effects of malnutrition are resolving; however, these medications should be considered when depression persists [F].

Patients who are persistent purgers should have ongoing monitoring of serum potassium levels. Chronic

hypokalemia should be treated with oral potassium supplementation [F].

5. *Discharge criteria for hospitalized patients.* Patients may be discharged from the hospital when they are medically stable and weight has been restored to a suitable level, behavioral symptoms have been substantially controlled, sufficient work with psychological and family factors has been undertaken assuring that aftercare treatment will be focused on relevant areas, and a targeted aftercare plan has been formulated and can be implemented.

6. *Longer-term goals.* Longer-term goals include improving enduring moods, personality disturbances, interpersonal relationships, and social functioning. Supportive and educative therapies should begin on initial contact. For patients accessible to insight therapy, a relationship with a therapist should be established as soon as possible [F].

7. *Family involvement.* Families should usually be engaged from the beginning of treatment and included in family meetings and treatment planning sessions [E,F]. Psychotherapy with the family (if possible with at least both parents, if not all family members) should be instituted when family members are able to participate without being persistently, destructively critical toward the patient and when no family member is so disruptive as to preclude productive work [F]. Hypercritical parents may first be seen without the patient present to help prepare them to participate in constructive family therapy that includes the patient. Family therapy is most useful for younger patients [A], and many experts consider family therapy to be mandatory for children and younger adolescents [F]. Marital therapy may be useful for married patients [F].

8. *Psychotherapy.* Psychotherapy should be tailored to the level of cognitive development, style, and complexity of the individual patient and family. Empathic support, education, insight, and problem solving should be used as soon as the patient is accessible to them. Because of the enduring and tenacious quality of many of the psychopathological and personality disturbance features and the need for considerable change and support during recovery, ongoing treatment in individual and/or group settings, at varying intervals depending on the patient's psychopathology and medical status, is frequently required for at least a year and often for several years [E,F].

9. *Considerations regarding chronicity.* Since many patients have a chronic course of illness, are unable to maintain a healthy weight, and often suffer from chronic depression, obsessionality, and social withdrawal, individualized treatment planning and careful case management are necessary. Treatment may require consultation with other specialists, subsequent rehospitalization, partial hospitalization, residential care, individual and/or group therapy, medications as indicated, and other social therapies [E,F]. Communication among professionals is important throughout outpatient care. With chronic patients, small progressive gains and fewer relapses may be the goals of psycho-

logical interventions. More frequent outpatient contact and other supports may sometimes help to prevent further hospitalization [E,F]. Expectations for weight gain with hospitalization may be more modest for chronic patients; achieving a safe weight rather than a healthy weight may be all that is possible.

Other options which may be recommended in individual circumstances:

1. *Dietary supplements.* Treatment options for nutritional rehabilitation include supplementation—and for treatment-resistant hospitalized patients, replacement—of regular food with liquid food supplements until the patient can return to normal table food [E]. Normal foods are best introduced as soon as possible to help the patient overcome “food phobias.”

2. *Enteral tube feedings and parenteral alimentation.* Rarely used options, in most instances requiring life-threatening or very unusual circumstances, may include nasogastric tube or in extreme cases parenteral feedings. These potentially life-saving interventions should be used for as brief a period of time as is necessary while normal eating is developed [E,F]. When the patient strongly objects to nasogastric or parenteral feeding in a life-threatening situation, an ethics consultation may be useful. Most consultants agree that these interventions should be used only when indicated by the patient’s medical condition and not as a means of behavioral manipulation, although some believe that in early renourishment some severely malnourished patients accept passive feeding via nasogastric tube more easily than the active choice of eating [F].

3. *Medications.* At some points during treatment, tricyclic antidepressants [A,E], cyproheptadine [A], fluoxetine [E], antipsychotics [A,E], and antianxiety agents [E] may be useful for some patients. Medications should not be used as the sole or primary treatment for anorexia nervosa.

4. *Management models.* Some programs routinely arrange for “split management” models of treatment, wherein a therapist primarily conducts the psychodynamic therapy and another psychiatrist writes orders, handles administrative and medical requirements, and works on changing the disturbed eating and weight patterns directly. For this split management model to work effectively, all personnel must work closely together, maintaining open communication and mutual respect to avoid reinforcing some patients’ tendencies to play staff off against one another, i.e., to “split” the staff [F].

An alternative split management approach is to have medical care providers (e.g., specialists in internal medicine, pediatrics, adolescent medicine) manage general medical issues, such as nutrition, weight gain, exercise, and eating patterns, while the psychiatric providers address psychiatric issues.

5. *Support groups.* Support groups led by professionals or advocacy organizations led by lay personnel that provide patients and their families with mutual sup-

port, advice, and education about eating disorders and their treatment may be of adjunctive benefit.

Treatment approaches not recommended:

Twelve-step-based programs or other approaches that focus exclusively on the need for abstinence without attending to nutritional considerations or behavioral deficits are not recommended as the sole initial treatment approach for anorexia nervosa [F]. The potential utility of these approaches in the *adjunctive* treatment of anorexia nervosa is an unsettled issue.

C. TREATMENT OF BULIMIA NERVOSA

The following are recommended:

1. *Initial treatment approach.* Patients with bulimia nervosa uncomplicated by the abuse of laxatives, alcohol or drug abuse, psychosis, suicidality, or major personality disturbances rarely require hospitalization and may achieve substantial symptom reduction with brief individual [A] or group psychotherapies [A,G]. Nutritional counseling [A], cognitive-behavioral therapy [A], and simple behavioral techniques such as planned meals and diary keeping [A] appear particularly helpful for initial symptom management, interrupting the binge-purge behaviors. With such approaches, some degree of clinical improvement is often evident within 2 to 4 months of treatment. Psychodynamically and interpersonally oriented psychotherapies [E,F] and psychoanalysis [E,F] often help such patients recognize and alleviate conflicts that contribute to their symptoms.

2. *Indications for hospitalization.* Indications for hospitalization include serious concurrent medical problems, psychiatric disturbances that would warrant the patient’s hospitalization independent of the eating disorder diagnosis, or severe and disabling symptoms (e.g., multiple daily binges and purges that significantly disrupt vocational performance or activities of daily living, unremitting laxative abuse) which have not responded to adequate trials of competent outpatient treatment [E,F]. In cases where other treatment options such as suitable partial hospitalization or residential programs are not available locally, hospital-based treatment may occasionally be provided initially for severely symptomatic patients [F].

3. *Discharge criteria for hospitalized patients.* Patients may be discharged from the hospital following substantial control of binge-purge cycles, laxative abuse, and other disabling symptoms, and when a targeted aftercare plan has been formulated and can be implemented.

4. *Antidepressant medications.* Antidepressant medications may reduce symptoms of binge eating and purging independent of the presence of depression [A]. Antidepressants may be used as one component of an initial treatment program for most patients, but should not constitute the entire treatment [A,C,E]. They may

be especially helpful for patients with significant symptoms of depression, anxiety, obsessions, or certain impulse disorder symptoms, or for patients who have failed previous attempts at appropriate psychosocial therapy [F]. Often, several different antidepressant medications may have to be tried sequentially to achieve the optimum effect. Doses of tricyclic and MAOI antidepressants for treating bulimia nervosa parallel those used to treat depression, although doses of fluoxetine higher than those used for depression may be more effective for bulimic symptoms [B,C,E,F]. In cases where symptoms do not respond to medication, it is important to assure that the patient has not taken the medication shortly before vomiting. Serum levels of medication may be obtained to determine whether presumably effective levels have actually been achieved [F].

5. *Psychotherapy.* Because of high rates of comorbid mood, anxiety, and personality disturbances and persistent, unresolved conflicts, recovering patients may achieve more lasting changes by continuing in extended psychotherapy or psychoanalysis that addresses relapse prevention and intrapsychic and interpersonal issues that come into focus as the initial symptoms of bulimia abate [C,E,F]. Psychodynamic, interpersonal, cognitive, or psychoanalytic approaches are most useful during this period. Therapeutic work may focus on common themes of development, identity formation, sexual and aggressive difficulties, affect regulation, gender role expectations, family dysfunction, coping styles, and problem solving. Patients with concurrent anorexia nervosa and/or concurrent borderline personality disorder usually require extended treatment [E,F].

6. *Family therapy.* Family therapy should be considered whenever possible and especially for adolescents

still living with their parents, older patients with ongoing conflicted interactions with parents, or patients with marital discord [E,F].

7. *Concurrent substance abuse disorders.* Unless malnutrition is severe, concurrent substance abuse disorders should usually be attended to first, since successful treatment for bulimia nervosa in the presence of an active substance abuse disorder is unlikely. Where treatment staff are competent to treat both disorders, concurrent treatment may be attempted [E,F].

Other options which may be recommended in individual circumstances:

Twelve-step programs such as Overeaters Anonymous may be helpful as an adjunct to initial treatment of bulimia nervosa and for subsequent relapse prevention [C,E]. Because of the great variability of knowledge, attitudes, beliefs, and practices from chapter to chapter and from sponsor to sponsor regarding eating disorders and their medical and psychotherapeutic treatment, and because of the great variability of patients' personality structures, clinical conditions, and susceptibility to potentially countertherapeutic practices, clinicians should carefully monitor patients' experiences with 12-step programs [F].

Treatment approaches not recommended:

Twelve-step-based programs or other approaches that exclusively focus on the need for abstinence without attending to nutritional considerations or behavioral deficits are not recommended as the sole initial treatment approach for bulimia nervosa [F].

IV. AREAS FOR FUTURE RESEARCH

The many gaps in our knowledge are evident, but several areas requiring considerable research stand out.

A. Biological, psychological, and social predictors of recovery and nonrecovery for treated and untreated anorexia nervosa and bulimia nervosa patients:

1. The impact of various comorbid conditions including mood, anxiety, substance abuse, personality, and other commonly encountered concurrent disorders on course and treatment response.

2. Modifications of treatment necessary in the presence of comorbid conditions.

B. Treatment evaluation and outcome studies that attend to patient preferences, long-term outcomes, and costs in relation to:

1. Incrementally intensive care programs ("stepped-care") and treatment package approaches.

2. Inpatient versus partial hospitalization programs for anorexia nervosa (to establish better criteria for defining appropriate durations for hospital and partial hospital care).

3. Newer biological agents affecting mood, anxiety, hunger, and satiety.

4. Psychodynamic, interpersonal, psychotherapeutic, and psychoanalytically based treatments.

5. Treatments based on and/or including 12-step and other recovery models.

6. Addressing the unique developmental (biological, psychological, and social) needs of children and adolescents with eating disorders.

7. Nutritional counseling strategies to successfully facilitate maintenance of healthy eating habits and weight in recovering patients.

8. Alternative treatment approaches.

9. Methods to facilitate early recognition and referral.

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VI. ORGANIZATIONS SUBMITTING COMMENTS

Academy of Psychosomatic Medicine
 Alcohol, Drug Abuse and Mental
 Health Administration
 American Academy of Child and Adolescent Psychiatry
 American Academy of Clinical Psychi-
 atrists
 American Academy of Family Physi-
 cians
 American Academy of Pediatrics
 American Academy of Psychiatrists in
 Alcoholism and Addiction
 American Academy of Psychiatry and
 the Law
 American Association of Chairmen of
 Departments of Psychiatry
 American Association of Community
 Psychiatrists

American Association of Psychiatric
 Administrators
 American Association of Psychiatric
 Services for Children
 American Association of Psychiatrists
 from India
 American Board of Forensic Psychia-
 try, Inc.
 American College of Neuropsychophar-
 macology
 American College of Physicians
 American College of Psychoanalysts
 American Hospital Association
 American Nurses Association
 American Psychoanalytic Association
 American Psychological Association
 American Society of Addiction Medi-
 cine

Anorexia Nervosa and Related Eating
 Disorders
 Group for the Advancement of Psychi-
 atry
 Joint Commission on Accreditation of
 Health Care Organizations
 National Association of Private Psychi-
 atric Hospitals
 National Association of Social Workers
 National Institute of Mental Health
 National Institute on Alcohol Abuse
 and Alcoholism
 National Institute on Drug Abuse
 National Mental Health Association
 Research Society on Alcoholism
 Society for Adolescent Medicine
 U.S. Veterans Administration, Mental
 Health Section

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Use of Posttraumatic Stress Disorder to Support an Insanity Defense

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Objective: The authors examine the allegation that the diagnosis of posttraumatic stress disorder (PTSD) is frequently abused in the legal system as the basis for a defense of not guilty by reason of insanity. **Method:** Data for the investigation were drawn from a study of insanity pleas gathered from court records in 49 counties in eight states. Data on the 28 insanity plea defendants for whom PTSD was diagnosed before or immediately after trial were compared with data on 8,135 defendants whose insanity pleas were based on other diagnoses. **Results:** Insanity pleas by defendants with diagnoses of PTSD constituted only 0.3% of the cases. There were few significant differences between the two groups on demographic variables, psychiatric histories, previous involvement in crime, or current charges. The defendants with PTSD were more likely to have been married, less likely to have been arrested as juveniles, and less likely to have been detained after trial. **Conclusions:** Contrary to previously expressed concerns, PTSD was infrequently associated with an insanity defense in the cases in this study. In the cases in which pleas based on PTSD were used, they were no more likely to succeed than pleas based on any other diagnosis. Defendants with PTSD-related insanity defenses differed little from other insanity defendants, contradicting the stereotype of the person who is driven by PTSD to commit crimes. The data do not support fears of widespread misuse of the diagnosis of PTSD in connection with the insanity defense.

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Posttraumatic stress disorder (PTSD) entered the lexicon of psychiatric diagnoses, DSM-III, in 1980 to cover a range of symptoms caused by "a recognizable stressor that would evoke significant symptoms of distress in almost everyone." This definition was modified in DSM-III-R in 1987 to cover those events that are

"outside the range of usual human experience and that would be markedly distressing to almost anyone." Although the concept of mental disorder resulting from psychological trauma is hardly a new one (1), its formal recognition as a distinct disorder in 1980 followed efforts by veterans' groups and those involved in the care of veterans to achieve recognition for a "post-Vietnam syndrome" (2). By October 1980, the Veterans Administration recognized PTSD as a potentially compensable disorder (3). An epidemiologic study of the general population identified PTSD in 1% of the sample, 3.5% of victims of physical attack, and about 20% of wounded veterans of the Vietnam war (2).

The potential usefulness of PTSD to the legal system was quickly recognized (4). In addition to its role as the underlying mental condition necessary for the insanity

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defense, PTSD has also been used to excuse or mitigate criminal responsibility in cases involving battered women (5, 6), automatism (7–10), the defense of diminished capacity (4, 11, 12), and the negation of specific (13–15) and general (16) criminal intent. PTSD as part of the factual background of a criminal case can affect the disposition of the case in other ways as well (17). In cases involving veterans, the largest impact of PTSD may well be in pretrial diversion, plea bargaining, and sentencing (4, 18). Proof that an alleged rape victim exhibits the symptoms of PTSD has been accepted in some courts as evidence demonstrating that the victim has indeed been raped (19).

The effect of PTSD on the legal landscape has not been confined to the criminal arena. Although it took longer to emerge as a factor in civil cases (beyond the obvious and immediate impact of the diagnosis on the awarding of veterans' benefits), the civil legal imagination has now discovered PTSD to such a degree that it was recently labeled by one court as the "diagnosis of choice" (20). PTSD and stress-related claims now account for 14% of all occupational disease claims (figures from the National Council on Compensation Insurance) and have had a profound impact on workers' compensation claims (21). Claims of discrimination and harassment in the workplace now regularly seem to include a cause of action or damages based on PTSD. PTSD has lent the authority of psychiatry to an increased range of emotional injuries and has entered the long-standing debate about whether bystander mental distress should be a compensable injury (22). Immigration lawyers and academics who specialize in refugee and asylum work now tout the use of forensic reports of PTSD to prove clients' well-established belief that they will be subjected to political persecution or harm if they are returned to their native countries (23). Evidence of PTSD in a child has been used as part of the basis for termination of parental rights (24).

There is great concern in both the legal and psychiatric communities that the tendency of advocates to push a mental disorder diagnosis to its outer limits to secure legal relief for their clients may pose a particular threat in the case of PTSD. Methods for objective assessment of the disorder are "relatively primitive" (25), since diagnosis of the condition almost always depends on self-report by the person seeking compensation, treatment, benefits, or a criminal defense or excuse. The danger of malingering is very real (26).

Furthermore, in the literature there is professional concern that the legal "potential" of PTSD will undercut the scientific rigor with which it is diagnosed by mental health professionals and with which legally relevant conclusions are drawn. Noting that a person who suffers from PTSD is not, for that reason alone, free of criminal responsibility, Mendelson (27) cautioned, "It is important that the diagnosis of PTSD not be brought into disrepute by becoming the basis of the insanity defense on the spurious grounds that it is a mental disease and thus that, automatically, all persons with the diagnosis are not responsible for their behavior." Sparr et

al. (18) urged that a careful distinction be drawn between PTSD leading to a dissociative state during a criminal act (which may be relevant to a claim of not guilty by reason of insanity) and other sequelae of combat experiences, since "otherwise, lawyers and forensic psychiatrists will continue to stretch and pull the diagnosis of PTSD well beyond its original intent in order to argue a criminal defense."

These concerns reflect a tension inherent in the forensic enterprise. Given the adversarial nature of our legal system, the push for certainty rather than complexity in fashioning forensic testimony, and the lack of any external control—beyond cross-examination—on the scientific validity of expert testimony, there is always the danger that what is testified to in court may exceed what has been demonstrated in the clinic or is contemplated by the DSMs (28). The problem appears to be particularly acute with something as new, as "unverifiable," as potentially useful, and as politically charged as PTSD. However, asserting that the problem exists falls short of demonstrating that this is so. To date there has been little evidence, beyond the anecdotal or the fears of professionals, upon which to base such a conclusion.

This article reports data that shed some light on the question of whether the diagnosis of PTSD is being "overused" in criminal cases involving the insanity defense. Although the data were not designed to test the use of PTSD *per se*, they nevertheless afford an opportunity to examine in the states surveyed the frequency with which PTSD was offered as an excusing condition in criminal cases based on the insanity defense and to look at the outcomes in those cases. These data cannot provide direct answers to the question of whether PTSD has been misrepresented or misused in court, but they may serve to suggest the dimensions of whatever problems exist.

METHOD

The data presented in this article were drawn from a study of the effects of insanity defense reforms conducted by Steadman and colleagues and described in detail elsewhere (29–31). Data were collected on insanity pleas in 49 counties in eight states (California, Georgia, Montana, New Jersey, New York, Ohio, Washington, and Wisconsin). These counties were selected to account for approximately two-thirds of insanity acquittals in each state. There was some variability in the years covered in each state, as demonstrated in table 1.

In an effort to identify a complete sample of cases in which an insanity plea was entered at any point in the criminal process, not just the cases that resulted in acquittal, records relating to nearly 1 million indictments were examined. Depending on the method of record keeping in each jurisdiction, this involved searches of court dockets, case files, competency-to-stand-trial referrals, and occasional computerized databases. The researchers found 8,953 insanity pleas. Data concerning each case were recorded on a standardized data

collection instrument. Follow-up information was obtained from records of mental health and corrections departments.

The analyses we report are based on the 8,163 cases collected from 1980 to 1986. Diagnoses were coded according to DSM-III or DSM-III-R nomenclature, with room for up to two diagnoses to be recorded for each evaluation the defendant received. Research assistants conducting the record reviews were instructed to record the first axis I diagnosis listed and either the first axis II diagnosis or, if there was no axis II diagnosis, the second axis I diagnosis. Since PTSD was not recognized as an official diagnosis prior to 1980, defendants whose cases were processed before that time who might have qualified for the diagnosis had been given diagnoses in other categories. Without data on specific symptoms, which were not collected, identification of those cases was not possible. Thus, we eliminated data collected prior to 1980 from this report.

Diagnostic data were potentially available for each defendant from three time periods: before trial, at post-trial hospital admission (if such hospitalization took place), and at posttrial hospital discharge. Because the study was not designed to focus on the use of particular diagnoses in the insanity defense, the data collection forms provided no opportunity for the data gatherers to indicate which diagnosis (when there was more than one) was relied upon as the basis for the insanity plea. Such information might have been difficult to obtain in a study based on review of records in any event.

The designation of a case as a PTSD-related insanity defense, therefore, depends on an inference concerning the co-occurrence of a PTSD diagnosis and an insanity plea. The inference—that the plea was based on the diagnosis of PTSD—is strongest when the diagnosis was made prior to trial ($N=22$). In 10 of these cases no other diagnosis was made. In eight cases the only other diagnosis was a nonpsychotic disorder. In four cases a psychotic or other major psychiatric disorder (e.g., major depressive disorder) was diagnosed.

The inference that PTSD was relied upon for an insanity defense is somewhat weaker—but may still be reasonable—when the diagnosis was recorded at the time of the hospital admission immediately following the trial ($N=6$). Weakest of all is the inference that might be drawn when the PTSD diagnosis was recorded only at the time of hospital discharge ($N=2$).

For the purpose of data analysis, therefore, two categories of PTSD-related insanity cases were used: those in which the diagnosis was made prior to trial ($N=22$) and those in which the diagnosis was made either prior to trial or at the time of the posttrial hospitalization ($N=28$). Cases in which the diagnosis was made only at the time of hospital discharge were not considered PTSD insanity cases. Comparison of the results for the two alternative definitions revealed no differences in the variables that were significantly associated with the use of a PTSD insanity plea, except for one variable (length of posttrial hospitalization) for which the small sample size ($N=5$ for the PTSD group) made conclu-

TABLE 1. Periods for Which Data Were Gathered on PTSD-Related Insanity Defense Pleas in Eight States

State	Study Years
California	July 1978 to June 1987
Georgia	January 1976 to December 1985
Montana	January 1976 to December 1985
New Jersey	January 1976 to December 1985
New York	October 1977 to September 1987
Ohio	January 1977 to December 1983
Washington	July 1979 to December 1987
Wisconsin	July 1979 to June 1985

sions problematic. Thus, the analyses presented here are based on the larger sample of PTSD-related insanity cases ($N=28$), defined by either pretrial or hospital admission diagnoses.

RESULTS

Of the 8,163 defendants pleading not guilty by reason of insanity whose cases were collected from 1980 to 1986, 28 (0.3%) had been given diagnoses of PTSD. Use of insanity pleas by defendants diagnosed as having PTSD fluctuated over time and across jurisdictions (table 2). PTSD diagnoses as a percentage of all insanity pleas in the jurisdictions studied peaked in 1983 at 0.7%. Wisconsin had the highest rate of PTSD insanity pleas (0.9%), and New Jersey, where no PTSD-associated pleas were recorded, had the lowest.

To ascertain whether defendants using a PTSD-associated insanity plea differed from other insanity plea defendants, the two groups were compared on multiple variables. The mean age of the PTSD defendants was 32.1 years ($SD=6.2$), and the mean age of the other defendants was 30.6 years ($SD=9.8$), a nonsignificant difference. There were no significant differences on most other demographic measures, including race, sex, and level of education (table 3). PTSD-related insanity defendants were significantly more likely to be married or to have been married previously. Unfortunately, data were not available on defendants' histories of military service.

With regard to past psychiatric and criminal histories, PTSD insanity defendants were significantly less likely to have been arrested as juveniles. There were no significant differences in the rate of previous psychiatric hospitalizations, arrests as adults, arrests for violent crimes, or previous prison stays.

The characteristics of the crimes with which the defendants were charged did not differ between the two groups. PTSD insanity defendants were not significantly more likely to be charged with crimes of violence, crimes perpetrated against males or females, or victimless crimes.

A few differences were found in the handling of the two groups by the criminal justice system. The PTSD insanity defendants were less likely to be found incompetent to stand trial. They were tried more often before

TABLE 2. Incidence of PTSD-Related Insanity Defense Pleas in Eight States in 1980-1986

Year	California	Georgia	Montana	New Jersey	New York	Ohio	Washington	Wisconsin	Total	Percentage of All Pleas
1980	0	0	1	0	0	1	0	0	2	0.1
1981	1	0	0	0	1	1	1	0	4	0.2
1982	1	0	0	0	0	4	0	1	6	0.4
1983	2	1	2	0	0	1	0	4	10	0.7
1984	0	0	0	0	1	2	1	0	4	0.4
1985	1	0	0	0	0	0	0	0	1	0.1
1986	0	0	—	0	1	0	0	0	1	0.3
Total	5	1	3	0	3	9	2	5	28	0.3
Percentage of all pleas	0.3	0.1	0.6	0.0	0.4	0.6	0.4	0.9	0.3	

TABLE 3. Data on Defendants With PTSD-Related Insanity Defense Pleas and All Other Defendants Pleading Not Guilty by Reason of Insanity in Eight States in 1980-1986

Item	Percentage of Defendants With PTSD (N=28) ^a	Percentage of All Other Defendants (N=8,135)
Demographic data		
White race	61.5	52.3
Male sex	85.7	89.4
High school graduate at least	65.4	49.7
Never married ^b	29.6	56.9
Past history		
One or more psychiatric hospitalizations	62.5	70.0
Arrested as a juvenile ^c	11.1	42.5
Arrested as an adult	72.0	74.8
Arrested for violent crime	45.8	40.6
Prison stay	41.7	35.1
Characteristics of offenses		
Violent or sexual crimes	63.2	69.3
Male victims	32.0	27.7
Female victims	32.0	28.8
Victimless crimes	36.0	40.9
Handling by criminal justice system		
Judged incompetent to stand trial	12.0	21.2
Jury trial	21.4	10.9
Verdict		
Not guilty	0.0	0.9
Guilty	60.7	46.9
Not guilty by reason of insanity	28.6	41.5
Guilty but mentally ill	3.6	3.2
Other	7.1	7.5
Disposition		
Incarceration	32.1	35.6
Hospitalization	21.4	39.0
Not detained ^d	44.4	23.5

^aNumbers on which percentages are based vary because of missing data on some items.

^bSignificant difference between groups ($\chi^2=8.3$, $df=1$, $p<0.01$).

^cSignificant difference between groups ($\chi^2=7.2$, $df=1$, $p<0.01$).

^dSignificant difference between groups ($\chi^2=6.5$, $df=1$, $p<0.05$).

a jury and were found guilty more frequently than the comparison group, but none of these differences reached statistical significance. However, the PTSD insanity defendants were significantly less likely to be de-

tained after trial, whether in a penal or a psychiatric facility, and more likely to be released on probation or some other status.

DISCUSSION

These data suggest that fears of widespread abuses related to the diagnosis of PTSD in the criminal courts are misplaced. In the first 6 years after the formal introduction of the diagnosis, PTSD appeared infrequently in insanity cases in the eight states studied. Indeed, in no state was the diagnosis of PTSD made in more than 1% of cases involving the insanity defense, and in one state it was entirely absent from the cases sampled.

These data do not allow us to rule out possible misuse of the diagnosis in the small number of insanity cases in which it was used, but there are some indications that this is unlikely. We hypothesized that a number of factors might be associated with malingering or fabricated insanity pleas based on PTSD: more serious charges (providing a stronger incentive to malingering), more sophistication about psychological symptoms (for which we used level of education as a measure), and more sophistication about the criminal justice system (as measured by past criminal charges as an adult). Although none of these variables is likely to be a perfect predictor of malingering or a fabricated diagnosis, it is noteworthy that none of them showed a significant relation to PTSD insanity pleas.

Moreover, the data suggest that measured by the acquittal rate, PTSD is not a particularly successful diagnostic category to use for claims of insanity. Defendants with PTSD were no more successful in their insanity pleas than were defendants given other diagnoses. In fact, a substantially (but not significantly) higher percentage of PTSD insanity defendants were found guilty on the charges against them.

With the caution required by the small number of cases in which PTSD was assigned as a diagnosis, the data can also be used to suggest the characteristics of defendants who use a PTSD-associated insanity defense. Clearly, PTSD insanity defendants are more like than unlike other defendants relying on an insanity plea. Their significantly higher rate of past or present

marriage, however, suggests greater stability in early adult life. This might be due to a later onset of illness than that for other diagnostic categories or a greater delay in the initiation of criminal behavior. The latter explanation is supported by the significantly lower rate of juvenile arrests in the PTSD group. In these ways, the data tend to confirm the popular perception of PTSD defendants.

Other differences that might be expected to occur between the two groups, however, on the basis of stereotypic images of PTSD insanity defendants, do not appear in our data. Such defendants are often pictured in the media as tortured but essentially upstanding veterans, who at some point become overwhelmed by their symptoms, "snap," and commit a violent crime. Belying this image, PTSD defendants in our sample were as likely as other defendants who used an insanity defense to have had previous involvement with the criminal justice system. The overall rate of previous arrests as adults for this group was substantial (72.0%), as were the rates of previous arrests for violent crimes (45.8%) and previous incarceration (41.7%). It is possible, of course, that some of these previous arrests might have resulted from behavior consequent to undiagnosed PTSD (especially before 1980); as already noted, data on defendants' status as veterans were not available.

If PTSD insanity defendants differed little from the rest of the sample at entry into the criminal justice system, there was a clearer distinction at the other end of the process. They were significantly less likely to be confined following adjudication, a result of lower rates of both imprisonment and hospitalization. From the perspective of the criminal defense bar, not to mention the defendants themselves, the potential for this kind of result more than justifies the effort involved in mounting a PTSD insanity defense. The differential impact of PTSD on the avoidance of prison or hospitalization after trial, with a greater reliance on probation, is particularly noteworthy given that the PTSD insanity defense group had a somewhat higher rate of conviction and no lower rate of past violence.

The lower frequency of hospitalization for PTSD insanity defense acquittees perhaps may be explained in part in terms of practicality. PTSD, unlike many psychotic disorders, can often be treated on an outpatient basis; and apart from specialized units found mainly in the Veterans Affairs system, most inpatient psychiatric programs probably have little to offer persons with PTSD. Taken together, the data on disposition support the view that once diagnosed, PTSD is particularly effective in helping defendants avoid posttrial detention.

Several caveats concerning these data should be kept in mind. The study from which they were drawn was designed for other purposes. It is possible, therefore, that not all diagnoses of PTSD were recorded, leading to some underestimation of incidence and a minimization of differences between the groups. Moreover, the inferential nature of the association between PTSD and use of an insanity defense is another reason for caution

in interpreting our results. Nonetheless, since these data are drawn from the largest study of the insanity defense ever performed, and one which is unlikely to be duplicated soon, they shed the best available light on the role of PTSD in the process.

There are also some issues that these data simply do not address. The data we analyzed were collected at the latest only through 1986. We cannot say that the use of PTSD in association with an insanity defense has not become more of an issue since that time, because of either the frequency with which it has been raised or a change in its acceptability to jurors. Nor can we speak to the possible incidence of the use of PTSD in mental state defenses not based on insanity. In fact, as we have noted, court decisions and law review articles reveal creative attempts to use PTSD in this way. Moreover, our data, which are focused entirely on the insanity defense, do not address the possible misuse of PTSD diagnoses in other criminal defenses or in civil cases, issues that are worth empirical investigation.

It does appear, however, that initial concerns about the role of PTSD in insanity defense proceedings were overblown, perhaps in keeping with general public skepticism about the insanity defense. As with other aspects of the insanity plea, the empirical data confirm that the PTSD diagnosis is more discussed than used.

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Childhood Physical Abuse and Combat-Related Posttraumatic Stress Disorder in Vietnam Veterans

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Objective: Early trauma in the form of childhood physical or sexual abuse has been associated with adult psychopathology. The purpose of this study was to compare rates of childhood abuse in Vietnam veterans with and without combat-related posttraumatic stress disorder (PTSD). **Method:** Premilitary stressful and traumatic events including childhood abuse and other potential predisposing factors were assessed in Vietnam combat veterans who sought treatment for PTSD (N=38) and Vietnam combat veterans without PTSD who sought treatment for medical disorders (N=28). Stressful and traumatic events including childhood physical abuse were assessed with the Checklist of Stressful and Traumatic Events and a clinician-administered interview for the assessment of childhood abuse. Level of combat exposure was measured with the Combat Exposure Scale. **Results:** Vietnam veterans with PTSD had higher rates of childhood physical abuse than Vietnam veterans without PTSD (26% versus 7%). The association between childhood abuse and PTSD persisted after controlling for the difference in level of combat exposure between the two groups. Patients with PTSD also had a significantly higher rate of total traumatic events before joining the military than patients without PTSD (mean=4.6, SD=4.5, versus mean=2.8, SD=2.9). **Conclusions:** These findings suggest that patients seeking treatment for combat-related PTSD have higher rates of childhood physical abuse than combat veterans without PTSD. Childhood physical abuse may be an antecedent to the development of combat-related PTSD in Vietnam combat veterans.

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The impact of premilitary risk factors on the development and severity of combat-related posttraumatic stress disorder (PTSD) has been a subject of interest since World War I. Although assessments of premilitary conflicts (1), personality (2), and neuroses (3) were used in early screenings of soldiers (4-6), these factors were not useful in the identification of individuals at risk for the development of PTSD (4). Premilitary personality continued to be investigated until the time of the Vietnam war (7-9), although these studies were limited by the lack of a systematic assessment of personality (10, 11).

Since the time of the Vietnam war there has been an increase in interest in premilitary risk factors for PTSD

(12-16). An association between age and years of education at the time of joining the service and the development of PTSD has been suggested (17). Family environment, childhood home life and stability, and social support before joining the military have not been associated with combat-related PTSD (18-21). Studies of premilitary antisocial behavior as a risk factor have reported conflicting findings (22, 23). Most studies have found that premilitary factors have not been significant predictors of PTSD (14, 15, 19). This has resulted in an increased emphasis on the importance of war-related traumatic stressors (6, 11, 17, 19, 24-33).

Several studies suggest an important relationship between childhood abuse and adult psychopathology (34-46). Although a number of potential premilitary risk factors have been examined, surprisingly, to our knowledge there have been no investigations of the relationship between combat-related PTSD and childhood abuse or childhood trauma in general. We designed the present study in order to determine if Vietnam veterans with combat-related PTSD have higher rates of childhood abuse, as well as childhood trauma in general, than Vietnam combat veterans without PTSD.

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TABLE 1. Premilitary Traumatic and Stressful Events in Vietnam Combat Veterans With and Without PTSD

Event	Patients With PTSD (N=38)		Comparison Subjects (N=28)		Odds Ratio	p
	N	%	N	%		
Physical and/or sexual abuse	11	29	2	7	5.30	0.03
Physical abuse	10	26	2	7	4.64	0.05
Sexual abuse	3	8	0	0		
Death of family member or close friend	18	49	15	54	0.78	n.s.
Life-threatening illness	8	21	5	18	1.23	n.s.
Exposure to dangerous fires	4	11	3	11	0.98	n.s.
Being rejected or put up for adoption	3	8	1	4	2.31	n.s.
Observing family violence	16	42	6	21	2.67	n.s.
Being the victim of armed robbery	6	16	4	14	1.13	n.s.
Having the house burglarized or car stolen	6	16	0	0	1.13	
Exposure to life-threatening natural disaster	6	16	9	32	0.40	n.s.
Joining the military before age 18	8	21	2	7	3.47	n.s.
Less than 12 years of education before joining the military	19	50	8	28	2.50	n.s.
Antisocial behavior in childhood	7	18	5	18	1.04	n.s.
Working in a stressful job (police officer, fire fighter)	2	5	1	4	1.50	n.s.

METHOD

The subjects were 66 Vietnam combat veterans at a Veterans Administration (VA) medical center. The veterans with PTSD (N=38) were seeking psychiatric treatment and included 25 of 27 subjects consecutively admitted to an inpatient PTSD unit during a 5-month period. An outpatient group with PTSD was included as a comparison group for potential response bias of an inpatient cohort. The outpatients consisted of 13 subjects consecutively admitted to the outpatient PTSD clinic during a 5-month period. Veterans without PTSD (combat comparison subjects) were seeking treatment for medical problems. This group consisted of 28 of 29 individuals who were physically able to participate in the study and were consecutively admitted to the outpatient ambulatory care clinic of a VA medical center during a 3-month period.

Patients and comparison subjects included in the study were those with a history of combat exposure. We defined combat exposure as having received hostile or friendly fire or having received incoming artillery rounds. Patients were assigned to groups with and without PTSD on the basis of a diagnosis of current

PTSD as measured by the Structured Clinical Interview for DSM-III (SCID) (47). Patients with a history of psychosis or organic brain syndrome were excluded from the study. Patients gave informed consent for participation in the study.

Premilitary traumatic and stressful events were evaluated with the Checklist of Stressful and Traumatic Events, with instructions to subjects to report only events that occurred before joining the military. The checklist is a 51-item self-report instrument with acceptable reliability and validity that assesses a broad range of traumatic events including childhood physical and sexual abuse (48; personal communication from J.L. Black, January 1989). Total number of traumatic events endorsed are summed to give a score for the checklist, which ranges from 0 to 51. Physical and sexual abuse are both assessed by a single global item of the checklist. In an attempt to confirm the accuracy of the checklist, patients and combat comparison subjects were evaluated with a structured interview designed to accurately document stressful and traumatic experiences. This interview was conducted by one of the investigators (J.D.B.), who was blind to the self-report data.

Other variables that have been suggested as potentially predisposing variables for the development of PTSD were evaluated, including childhood antisocial behavior (Helzer index) (22) and age and years of education at the time of joining the military. The Helzer index assesses truancy, criminal behavior, and other antisocial behavior before the age of 15.

In order to compare combat-related PTSD symptoms in patients with PTSD with and without a history of abuse, patients were evaluated with the Mississippi Scale for Combat-Related Posttraumatic Stress Disorder, a self-report measurement of current PTSD symptom severity with acceptable reliability and validity (49). The Brief Symptom Inventory-Global Index is an instrument for the evaluation of general psychiatric symptoms and has acceptable reliability and validity (50). Level of combat exposure was evaluated with the Combat Exposure Scale, another validated and reliable self-report instrument for the measurement of level of exposure to combat in Vietnam (51). High levels of combat exposure were defined as scores greater than 24 on the scale (personal communication, T.M. Keane, October 1991). Current drug and alcohol abuse were assessed with the Addiction Severity Index, a validated, clinician-administered instrument designed for the quantification of current problems with drugs and alcohol at the time of presentation for treatment (52).

Data from the Checklist of Stressful and Traumatic Events were analyzed with standard parametric statistical methods. Logistic regressions were performed in order to evaluate potential risk factors for the development of PTSD (outlined in table 1). Two-tailed tests of significance were used throughout, and Fisher's exact tests were used when there were less than five observations in a single cell. Significance was defined as $p < 0.05$.

RESULTS

There were no significant differences in demographic variables between the groups with and without PTSD. Patients with PTSD and comparison subjects were similar in age (mean=47.3 years, SD=2.7, versus mean=46.6, SD=2.8), years of education (mean=12.4, SD=1.7, versus mean=12.8, SD=1.8), age at the time of joining the military (mean=18.5 years, SD=1.1, versus mean=19.0, SD=1.9), and number of months spent in Vietnam (mean=15.3, SD=6.2, versus mean=14.3, SD=8.5). Patients with PTSD were similar in race (8% black, 3% Hispanic, 89% white) to comparison subjects (15% black, 7% Hispanic, 78% white). When marital status was considered overall, there was no significant difference between PTSD subjects (45% married, 8% separated, 39% divorced, 8% never married) and comparison subjects (32% married, 4% separated, 29% divorced, 35% never married), although PTSD subjects showed a greater tendency to have been married at some time in their life.

Patients with PTSD reported higher rates of childhood physical abuse, as measured by the self-report global assessment of physical abuse item on the Checklist of Stressful and Traumatic Events, than patients without PTSD (26% versus 7%) (table 1). The rates of self-reported sexual abuse were 8% versus 0%. In addition, there was no difference in rates of physical and/or sexual abuse between inpatients (28%, N=7 of 25) and outpatients with PTSD (31%, N=4 of 13).

The self-report global assessment of childhood physical abuse with the Checklist of Stressful and Traumatic Events was validated with a clinician-administered structured interview for stressful and traumatic events. Twelve patients were determined to have experienced physical abuse according to both the self-report and the structured interview. Two patients were assessed by the structured interview to have had physical abuse but did not report abuse on the Checklist of Stressful and Traumatic Events. One patient who was not assessed by the structured interview to have had abuse reported abuse on the checklist. The self-report checklist assessment of abuse was used for the classification of abuse as the more conservative of the two methods of assessment. There were no significant differences between the two groups in any of the other self-reported individual stressful and potentially predisposing premilitary events measured by the Checklist of Stressful and Traumatic Events (table 1). Patients with PTSD experienced a greater number of total traumatic events before joining the military, as measured by the checklist (mean=4.6, SD=4.5, versus mean=2.8, SD=2.9; $t=2.03$, $df=64$, $p<0.05$).

More patients with PTSD had high levels of combat exposure, as defined by a score of 24 or greater on the Combat Exposure Scale, than patients without PTSD (89%, N=34, versus 43%, N=12; $\chi^2=14.11$, $df=1$, $p<0.0001$). After controlling for the effects of combat exposure in a logistic regression model, there continued to be a significant association between exposure to child-

hood abuse and the development of PTSD (odds ratio=9.39, $p=0.04$).

The PTSD patient group was also examined alone to determine if there were differences between patients with and without a history of childhood abuse. There were no differences between these two subgroups in PTSD symptoms (Mississippi Scale for Combat-Related Posttraumatic Stress Disorder; mean score=134.6, SD=12.1, versus mean=130.7, SD=14.0), nonspecific psychiatric symptoms (Brief Symptom Inventory-Global Index; mean=2.32, SD=0.54, versus mean=2.27, SD=0.76), or combat exposure (Combat Exposure Scale; mean=29.1, SD=7.0, versus mean=33.5, SD=5.5). Abused patients with PTSD, however, were more likely to have witnessed family violence in childhood (mean=0.73, SD=0.47, versus mean=0.30, SD=0.46; $p=0.01$), showed more antisocial behavior in childhood (mean=2.91, SD=2.2, versus mean=1.26, SD=1.3; $p=0.03$), and had higher total scores on the Checklist of Stressful and Traumatic Events (mean=7.09, SD=4.97, versus mean=3.63, SD=3.90; $p=0.03$) than nonabused patients with PTSD.

DISCUSSION

Vietnam combat veterans with PTSD in our study had higher rates of childhood physical abuse than Vietnam combat veterans without PTSD. A history of childhood abuse was associated with combat-related PTSD after controlling for differences in level of combat exposure between the groups with and without PTSD. Patients with PTSD had a higher rate of total childhood traumatic events, as measured by the total score on the Checklist of Stressful and Traumatic Events, than patients without PTSD. There were no differences in other premilitary stressful or potentially predisposing risk factors measured in this study between the groups with and without PTSD (table 1). There were no differences in psychiatric symptoms, PTSD symptoms, or demographic variables between patients with PTSD with and without a history of childhood abuse.

It should not be concluded from this study that childhood physical abuse causes combat-related PTSD. Our small group size prevents generalizations from our results to other population samples. In addition, 74% of our patients with PTSD did not report a history of childhood physical abuse. It is also possible that our results may be secondary to different recall of early trauma between the two groups. We addressed the problem of recall bias by attempting to validate the self-report Checklist of Stressful and Traumatic Events with a clinician-administered structured interview that assesses abuse and other stressful and traumatic events and found an adequate correlation between the two methods of assessment.

Patients presenting for treatment of combat-related PTSD may, in fact, have PTSD symptoms secondary to noncombat trauma such as childhood abuse. Presumably, these veterans would be more likely to underre-

port childhood trauma in an attempt to establish combat trauma as the cause of their PTSD. Prospective longitudinal studies are needed to answer these questions about childhood versus combat-related trauma.

Patients with a history of abuse may be vulnerable to the development of combat-related PTSD. Individuals abused in childhood may have acquired characteristic methods of coping with stressful experiences, such as emotional numbing, which may, in fact, make them more susceptible to subsequent trauma such as combat stress. Studies from the Israeli wars suggest that combat veterans who developed acute stress reactions during combat were more likely to develop subsequent stress reactions during subsequent wars than were veterans with no history of stress reactions (53). These findings, in conjunction with our own results, provide evidence for the stress vulnerability rather than the stress inoculation hypothesis of trauma. In other words, exposure to stress early in life increases the vulnerability to psychopathology in response to subsequent stressors, rather than having a protective effect. Studies of the neurobiology of stress suggest that exposure to stress early in life may result in long-term changes in neurobiological systems that are involved in the stress response (54–58). The development of valid and reliable instruments for the assessment of the full range of childhood abuse experiences will help answer questions about the relationship between childhood trauma and the development of combat-related PTSD. This information could have implications for the prevention and treatment of PTSD.

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Psychopathology and Psychiatric Diagnoses of World War II Pacific Theater Prisoner of War Survivors and Combat Veterans

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***Objective:** This study assessed current and long-term psychological and psychiatric sequelae of war trauma in World War II Pacific theater combat veterans, some of whom had been Japanese prisoners of war (POWs). **Method:** A group of 36 POW survivors and a group of 29 combat veterans, all of whom had seen fierce fighting and heavy unit casualties, were compared approximately 40 years later on psychological instruments assessing psychopathology constructs, negative mood states, and symptoms of posttraumatic stress disorder (PTSD) and on the computer-administered National Institute of Mental Health Diagnostic Interview Schedule. **Results:** Although similar in personal backgrounds and in having suffered catastrophic war trauma, the two groups differed in the severity and type of psychiatric symptoms and in the occurrence of psychiatric disorders. Anxiety and depressive disorders were common in both groups, but there were differences in the frequency of PTSD diagnoses. Among the POW survivors, 70% fulfilled the criteria for a current diagnosis and 78% for a lifetime diagnosis of PTSD, compared to 18% and 29%, respectively, of the combat veterans. **Conclusions:** The findings point to the persistent nature of symptoms thought to be residuals of extraordinary stress and the relation between severity of psychiatric sequelae and characteristics of the stressors.*

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The physical and psychological abuse meted out to captives of the Japanese in the Pacific theater of World War II has been documented by firsthand observations (1), clinical descriptions (2, 3), and historical accounts (4, 5). These materials point to the horrendous brutality and persistent atrocities that led to the death of over 11,000 American military personnel and the bare survival of 16,358 prisoners of war (POWs) liberated after more than 3 years of captivity. Japanese forces seized Americans in China, Guam, and Wake Island in December 1941 and captured approximately 25,580 men and more than 50 women after the surrender of Bataan and Corregidor in April and May 1942 (3, 6). Rescue teams liberated Americans from Japan, Manchuria, Korea, Thailand, the Philippines, and the Celebes and learned of the terrorizing executions, unending semistarvation, and persistent psychological debasement and torture that constituted the Pacific theater POW experience in its entirety.

Clinicians and researchers have explored personality factors that led to survival under these horrifying conditions (1) and studied prisoners' psychological reactions to the Japanese POW experience immediately after their release (3). Over the past 40 years, however, most researchers have attempted to identify long-term medical and psychological residuals of war captivity. As an example, Canadian investigators documented impairments in nervous system and psychological functioning among Hong Kong POW survivors 20 years after liberation (7) and greater dysfunction in neuropsychological, psychiatric, and physical/neurological domains among Japanese-held former World War II POWs than among those who were held by the Germans (8). These findings have been confirmed by more comprehensive studies of medical and psychiatric outcomes among American former POWs.

In a follow-up of World War II and Korean conflict POW survivors over a 20-year period, Beebe (9) found that somatic sequelae were common among all former POW groups in the early years after liberation but described increased risks of hospitalization and psychiatric conditions particularly among Pacific theater POW survivors. Excess morbidity was shown to correlate with retrospective accounts of weight loss and nutritional deficiency diseases during confinement, and

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Keehn (10) reported that Pacific theater POW survivors were at greater risk of dying, specifically from suicide and tuberculosis. More recent studies have assessed symptoms of posttraumatic stress disorder (PTSD) (11, 12) and depression (13) and pointed to physical illness sequelae such as gastrointestinal morbidity (14), stryngyloidiasis (15), peripheral neuropathy (16), and beriberi (17).

Findings from this research revealed impairments in psychological functioning and an increased prevalence of psychopathology among Pacific theater POW survivors, but knowledge of specific psychiatric disorders and symptoms that may constitute long-term response to this extraordinary trauma is limited. For example, most investigators have not compared Japanese-held POW survivors with appropriate veteran control groups and applied multimethod psychopathology assessment sufficiently broad to explore both psychiatric diagnoses and specific stress-related symptoms. The present study assessed dimensions of psychopathology—including negative mood states such as anxiety and depression, specific symptom components of PTSD, and prevalence of current and lifetime psychiatric disorders—that might represent long-term residuals of Pacific theater POW confinement. We used demographic and personal history questionnaires, objective measures of psychopathology, a computer-administered diagnostic interview, and structured clinical interviews for the diagnosis of PTSD to compare a group of Pacific theater POW survivors and a group of combat veterans assigned to similar military duty.

METHOD

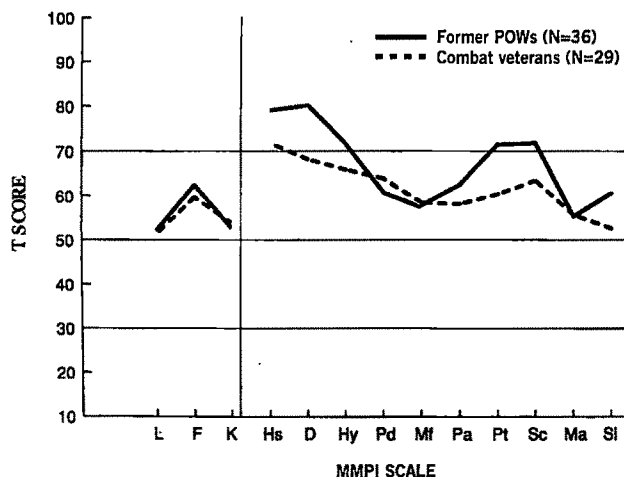
The participants were 44 World War II Pacific theater veterans who had survived POW confinement and 33 veterans who had seen heavy combat but had not been captured by enemy forces. The former POWs responded to a Department of Veterans Affairs (VA) invitation to undergo medical evaluation and completed the psychological and psychiatric assessment for this study as well. This number represented all Pacific theater POW survivors seen at our VA medical center over a 4-year period; however, data on eight of the men were excluded from the data analysis because they showed evidence of organic brain dysfunction ($N=4$), severe emotional distress ($N=3$), and active alcoholism ($N=1$). Thus, the final study group consisted of 36 former POWs. The combat veterans were recruited by letters inviting participation in a psychiatric assessment research protocol which were mailed to veterans randomly selected from among the medical center patients and to commanders of American Legion and Veterans of Foreign Wars posts. Data on four of these men were excluded from the data analysis because they showed evidence of organic brain impairment; this resulted in a final study group of 29 combat veterans.

The POW survivors were older than the combat veterans (mean age=67.03 years, $SD=3.00$, and mean=

64.83 years, $SD=2.90$, respectively; $F=8.89$, $df=1, 63$, $p<0.01$). There were no statistically significant differences between the POW group and the combat veterans in race (94% and 90% white, respectively), years of formal schooling (mean=13.06, $SD=3.00$, and mean=11.97, $SD=2.99$), cognitive sophistication as measured by the WAIS-R full-scale score (18) (mean=106.44, $SD=13.90$, and mean=101.38, $SD=11.95$), socioeconomic status as defined by Hollingshead-Redlich criteria (19) (75% and 76% middle to lower-middle class), military rank (92% and 97% enlisted men), marital status (86% and 79% married), and employment status (78% and 90% employed or voluntarily retired; 22% and 10% medically disabled). The former POWs were captured at a young age (mean=22.06 years, $SD=2.75$) after serving an average of 4 months in heavy combat, and 69% were wounded prior to or at the time of capture. They were interned for an average of 38 months and sustained an average confinement weight loss of 35%. The majority ($N=17$) were captured on Bataan and endured the death march. Others were taken prisoner at Corregidor ($N=11$), Wake Island ($N=3$), the Java Straits ($N=2$), Guam ($N=1$), Cuyo Island in the Philippines ($N=1$), and Kyushu, Japan ($N=1$). The combat veterans averaged 19 years of age at entry into the war and experienced roughly 16 months of combat marked by heavy casualties and injuries (41% wounded).

The assessment incorporated a multimethod strategy for measuring psychopathology constructs—such as anxiety and depressive features cited as common among Pacific theater POW survivors (11), current and lifetime diagnoses of mental disorders, and behavioral and emotional symptoms listed in DSM-III-R as characteristic of PTSD—against a backdrop of salient premilitary and postmilitary life events and circumstances. The psychological instruments included the WAIS-R, the MMPI (20), the Beck Depression Inventory (21), and the trait anxiety scale of the State-Trait Anxiety Inventory (22). A structured questionnaire was administered to obtain information on premilitary background and adjustment factors, childhood and adolescent emotional or behavioral problems, combat and POW confinement experiences, and work and family history subsequent to war duty. The computerized version (23) of the National Institute of Mental Health Diagnostic Interview Schedule (DIS) (24) was used to identify psychiatric disorders with the exception of PTSD. Symptoms of PTSD were assessed by the PTSD portion of the DIS structured interview and an experimenter-adapted, DSM-III-R-derived PTSD symptom checklist administered by a psychologist or psychiatrist.

Analysis of continuous variables applied an omnibus significance test to protect against inflation of the alpha level, as recommended by Cliff (25). Separate multivariate analyses of variance with a single group factor were performed for the MMPI scores (13 validity and clinical scales and supplemental scales for anxiety [26] and PTSD [27]) and the mood state measures (Beck inventory and trait anxiety scale), followed by corresponding

FIGURE 1. MMPI Profile Patterns of World War II Pacific Theater POW Survivors and Combat Veterans

univariate analyses. Multivariate analyses of covariance showed no significant effects for age, and only results of analyses of variance are reported. Categorical variables were examined using chi-square analyses with the highly conservative Yates correction.

DIS diagnoses were recorded and analyzed with the use of strategies applied in the national survey of the Vietnam war generation (28). Full data were available on 23 of the POW survivors and 28 of the combat comparison veterans. The former POWs were distressed and tended to terminate early, even though they were encouraged by the examiners who supervised the procedure to progress through all the DIS components. In addition to PTSD, the following disorders were examined: affective disorders, consisting of major depressive episodes, manic episodes, and dysthymia; anxiety disorders, including generalized anxiety disorder, panic disorder, and obsessive-compulsive disorder; substance abuse disorders, consisting of alcohol abuse and dependence combined into a single classification and drug abuse and dependence combined into a single classification; and antisocial personality disorder.

RESULTS

Similar in personal background characteristics, the former POW and combat comparison groups described stable emotional and behavioral adjustment during the developmental years. School suspensions or expulsions (6%) and truancy (17%) were reported with low frequency, and none of the men admitted substance abuse, arrests as adults, employment difficulties, psychiatric care, or suicide attempts prior to military service. Men in both groups reported having been assigned to combat duty in areas where fighting was fierce and claimed many lives. All study participants had been subjected to circumstances that constituted extraordinary stress and thus were viewed as meeting DSM-III-R criterion A for

PTSD (having experienced severely traumatic events sufficient to evoke stress-related psychopathology in almost anyone). The participants were not a treatment-seeking group, and only 28% of the POW survivors and 24% of the combat veterans had sought mental health treatment before the present assessment.

The former POWs described being captured after days of battle siege, heavy casualties, grossly limited food and ammunition supplies, and the stark realization that capture and death constituted predictable outcomes. Capture and confinement experiences included merciless death marches, capricious executions, and chronic frustration of bodily needs. The majority of the men reported psychological intimidation (97%), severe beatings (94%), semistarvation and malnutrition diseases (92%), unprotected exposure to extreme temperatures in the Philippines, Japan, and Manchuria (89%), overcrowding and unsanitary conditions leading to other medical illnesses (89%), witnessing senseless torture and gruesome executions (89%), incessant death threats (86%), and forced marches (83%). They also described coming under Allied fire (69%), deliberate physical torture (61%), interrogations (58%), personal death threats (56%), being wounded at capture (39%), and solitary confinement (31%).

Results of the MMPI and other objective tests revealed that the POW survivors showed more severe emotional distress in general, as well as higher scores on certain constructs viewed as fundamental to the psychopathology of stress-related mental disorders. The POW survivors and combat veterans differed on the 13 MMPI validity and clinical scales and the two supplemental scales overall ($F=1.92$, $df=15, 49$, $p<0.05$). As can be seen in figure 1, the former POWs produced more elevated profile patterns and significantly higher scores on scales D (depression; $F=10.27$, $p<0.01$), Pt (psychasthenia; $F=9.64$, $p<0.01$), Sc (schizophrenia; $F=4.93$, $p<0.05$), and Si (social introversion; $F=9.17$, $p<0.01$) ($df=1, 63$ for all analyses). They also scored higher on the MMPI PTSD scale (mean=17.42, $SD=10.73$, versus 11.76, $SD=9.21$; $F=5.06$, $p<0.05$). The groups did not differ on mood state measures overall (Beck Depression Inventory and trait anxiety scale), but the former POWs produced higher scores on the Beck inventory (mean=13.83, $SD=9.85$, versus mean=8.83, $SD=8.51$; $F=4.68$, $p<0.05$) and roughly equivalent scores on the trait anxiety scale (mean=43.17, $SD=13.68$, versus mean=37.00, $SD=10.83$).

The groups differed in the assignment of any current psychiatric diagnoses (74% for the former POWs and 32% for the combat veterans; $\chi^2=7.22$, $df=1$, $p<0.01$) but not of any lifetime diagnoses (83% and 64%, respectively). A list of the specific diagnoses identified in both groups is presented in table 1, which shows that the most frequently occurring disorders were those marked by anxiety and depressive features. The groups differed significantly only in current and lifetime diagnoses of PTSD: 70% of the POW survivors and 18% of the combat veterans met the criteria for current PTSD, and 78% of the former POWs and 29%

TABLE 1. Current and Lifetime Psychiatric Diagnoses^a of World War II Pacific Theater POW Survivors and Combat Veterans

Diagnosis	POW Survivors (N=23)				Combat Veterans (N=28)				χ^2 (df=1)	
	Current		Lifetime		Current		Lifetime		Current	Lifetime
	N	%	N	%	N	%	N	%		
PTSD	16	70	18	78	5	18	8	29	11.89 ^b	10.57 ^b
Generalized anxiety disorder	4	17	8	35	3	11	10	36	0.08	0.00
Dysthymia	3	13	4	17	3	11	4	14	0.00	0.00
Major depression	3	13	3	13	3	11	4	14	0.00	0.00
Obsessive-compulsive disorder	2	9	2	9	2	7	2	7	0.00	0.00
Panic disorder	1	4	1	4	0	0	2	7	0.01	0.00
Alcohol abuse/dependence	0	0	4	17	0	0	12	43	0.00	2.71
Manic episode	0	0	0	0	0	0	0	0	0.00	0.00
Antisocial personality	0	0	0	0	0	0	0	0	0.00	0.00
Drug abuse/dependence	0	0	0	0	0	0	0	0	0.00	0.00
PTSD comorbidity										
With other anxiety disorders	6	26	9	39	2	7	7	25	2.14	0.61
With affective disorders	4	17	4	17	5	18	6	21	0.00	0.00
With alcohol abuse/dependence	0	0	4	17	0	0	4	14	0.00	0.00

^aAccording to the National Institute of Mental Health Diagnostic Interview Schedule.^b $p < 0.01$.

of the combat veterans met the criteria at some point in their lives. The combat veterans were assigned lifetime alcohol abuse and dependence labels more often (43% versus 17%), but this trend was not statistically significant. The data presented in table 1 reflect high comorbidities among lifetime PTSD, other anxiety disorders, and affective disorders.

Stress-related symptoms, ordered by frequency of endorsement on the DIS PTSD and DSM-III-R-adapted PTSD interviews are outlined in table 2. High percentages of both veteran groups described the hallmark symptoms of PTSD: more than one-half of both groups reported disrupted sleep, recurrent intrusive recollections of traumatic events, irritability or outbursts of anger, and problems with concentration. As many as 13 of the possible 17 PTSD symptoms were endorsed by more than one-half of the POW survivors, and two-thirds of the combat veterans cited problems associated with sleep disturbances and persistent, intrusive, and distressing memories of trauma. Despite these similarities, the study groups differed significantly in the frequencies of endorsement of restricted range of affect, recurrent distressing dreams of the trauma, diminished interest in usual activities, interpersonal avoidance, physiological reactivity and psychological distress upon exposure to trauma-related events, sense of a foreshortened future, and exaggerated startle response.

DISCUSSION

Our findings are consistent with the trend toward identifying PTSD as a highly predictable, rather than remote, and persistent outcome of war imprisonment and war atrocities. Goldstein et al. (11) found current evidence of the full PTSD syndrome in roughly 50% of a group of Pacific theater POW survivors and a partial symptom picture in most of the rest of the group. Kluznik et al. (29) reported that 67% of European and

TABLE 2. Symptoms Endorsed^a by World War II Pacific Theater POW Survivors and Combat Veterans

Symptom	POW Survivors (N=36)		Combat Veterans (N=29)		χ^2 (df=1)
	N	%	N	%	
Sleep disturbance	32	89	22	76	1.12
Recurrent intrusive recollections	30	83	19	66	1.87
Irritability/anger outbursts	29	81	16	55	3.74
Feelings of detachment or estrangement	28	78	13	45	6.14 ^b
Concentration difficulties	28	78	15	52	3.78
Recurrent distressing dreams	24	67	9	31	6.80 ^c
Avoidance of activities or situations arousing recollections of the trauma	24	67	12	41	3.20
Loss of interest in usual activities	24	67	9	31	6.80 ^c
Restricted range of affect	23	64	7	24	8.68 ^c
Physiological reactivity upon exposure to trauma-related events	23	64	9	31	5.68 ^b
Exaggerated startle response	22	61	9	31	4.68 ^b
Intense psychological distress upon exposure to trauma-related events	21	58	8	28	4.96 ^b
Avoidance of thoughts or feelings associated with the trauma	21	58	9	31	3.78
Hypervigilance	15	42	6	21	2.34
Inability to recall trauma events	12	33	4	14	2.34
Sense of a foreshortened future	12	33	2	7	5.17 ^b
Reexperiencing the trauma	9	25	3	10	1.42

^aOn the PTSD portion of the National Institute of Mental Health Diagnostic Interview Schedule and in clinical interviews in which checklists of PTSD symptoms derived from DSM-III-R were used.^b $p < 0.05$.^c $p < 0.01$.

Pacific theater former POWs met criteria for a lifetime PTSD diagnosis, and Sutker et al. (30) identified current PTSD in 86% of a group of Korean conflict POW survivors. The finding of the present study that 70% of the Pacific theater POW survivors displayed the full symp-

tom picture for current PTSD complements these earlier reports and attests to the unremitting nature of stress-related psychopathology in substantial proportions of persons exposed to war catastrophe. Such high rates of PTSD four decades after termination of the stressor are relevant to projections of outcome for victims of contemporary war and political trauma. For example, Carlson and Rosser-Hogan (31) found a current PTSD rate of 86% among Cambodian refugees who were imprisoned in labor camps, tormented by physical abuse and outright torture, and deprived of property and human rights.

Whether taken prisoner or not, 40-year survivors of World War II Pacific theater trauma were characterized by psychopathology presumed in many instances to be stress-related. More than three-fourths of the former POWs and more than one-half of the combat veterans exhibited at least one lifetime mental disorder, with a preponderance of anxiety and depressive disorders. The prevalences of current and lifetime PTSD diagnoses and psychological symptoms were greater among the veterans who had been subjected to the most severe trauma, POW confinement, underscoring the "dose-dependent" relationship between psychiatric symptoms and severity of trauma that has been described in other research (32, 33). Assessment revealed similar constellations of symptoms in both veteran groups, including anxiety and negative ruminations, feelings of dysphoria, somatic concerns, and specific problems with sleep, recurrent traumatic memories, irritability, and lapses in concentration. However, psychopathology was more exaggerated among the former POWs, who showed greater symptoms of clinical depression, anxiety, worry, and interpersonal distancing evidenced by higher scores on MMPI scales D, Pt, Sc, Si, and PTSD, and the Beck measure of depression. Such elevated levels of psychopathology may derive in complex fashion from the experience of severe trauma as well as the chronic residuals of emotional and interpersonal maladjustment, further amplifying symptom expression and psychological distress over time.

The results of assessments of psychiatric disorders by both objective instruments and clinical interviews revealed that symptoms of anxiety and depression, intrusive recollections of traumatic events, and interpersonal avoidance and numbing phenomena were characteristic of veterans who suffered prolonged war imprisonment and were not necessarily uncommon among their counterparts who survived months of heavy combat without imprisonment. It could be argued that since both groups consisted of aging men, much of their psychopathology can be attributed to the effects of the increased physical disabilities and psychological vulnerabilities of aging. If this were the case, both groups might have been characterized more specifically and exclusively by symptoms of dysphoria and somatic preoccupations rather than anxiety, avoidance, and reexperiencing phenomena. It is also worth comment that despite the relatively high rates of psychopathology among the two groups, neither group was predomi-

nantly composed of men seeking psychiatric help, who might be accustomed to reporting psychological symptoms in detail for treatment purposes. Therefore, we interpret the findings as shedding light on the nature of PTSD, which consists of stress-related phenomena that are unremitting with the passage of time, variable in severity depending upon the characteristics of the stressors, and potentially exacerbated by the vicissitudes associated with aging.

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Lifetime Prevalence of Panic States

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Objective: Little is known about the prevalence of panic symptoms that do not meet criteria for panic disorder. This study was conducted to determine the prevalence of panic disorder, panic attacks, and limited-symptom attacks in the general population. **Method:** The authors identified a community-based sample of 1,683 randomly selected adults in 18 census tracts in San Antonio, Tex.; 1,306 of these subjects agreed to be interviewed with the Structured Clinical Interview for DSM-III. Subjects were classified as having panic disorder if they met DSM-III-R criteria, as having panic attacks if they had attacks of four or more panic symptoms but did not have panic disorder, and as having limited-symptom attacks if they had attacks of fewer than four symptoms but no full-blown panic attacks. **Results:** The crude lifetime prevalence rates were 3.8% for panic disorder, 5.6% for panic attacks, and 2.2% for limited symptom attacks. Women had higher rates of panic disorder and panic attacks than men, but the difference between men and women was not statistically significant for limited-symptom attacks. No statistically significant differences in rates between Hispanic and either non-Hispanic white or black subjects were found. Non-Hispanic white subjects had higher rates of limited-symptom attacks than black subjects. **Conclusions:** The prevalence of limited-symptom attacks in this community-based study was 2.2%; black subjects had lower rates than non-Hispanic white subjects. Panic attacks appear to be at least as common as DSM-III-R panic disorder and, like panic disorder, are more common among women.

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In the 13 years since the definition of panic disorder appeared in DSM-III, our understanding of the panic experience, its treatment, and its consequences has improved dramatically. However, DSM-III-R and preliminary discussion of DSM-IV raise the issue of subthreshold disorders and their significance, which presents a challenge to those developing diagnostic criteria for panic states. There is evidence that infrequent panic attacks—fewer than three in a 3-week period—involve a similar risk of phobic avoidance (1-4) and depression (3, 5) as do frequent attacks. Similarly, there appears to be little difference in the risk of phobic avoidance (1, 2) between limited-symptom attacks (those involving fewer than four panic-related symptoms) and full-blown attacks.

To date, most population-based prevalence data have focused on DSM-III-defined panic disorder. Little is known about the prevalence of panic attacks, and even

less is known about the prevalence of limited-symptom attacks. The studies that have been performed have generally used the National Institute of Mental Health Diagnostic Interview Schedule (DIS), for which concern about reliability and sensitivity exists, especially in the area of panic disorder (6).

We conducted the current study to determine the prevalence in the general population of DSM-III-R-defined panic disorder, panic attacks, and limited-symptom attacks, using the panic disorder portion of the Structured Clinical Interview for DSM-III (SCID) (7) in a community with a large Hispanic population.

METHOD

The Panic Attack Care-Seeking Threshold study was conducted in San Antonio, Tex., according to methods similar to those of the Epidemiologic Catchment Area (ECA) study (8) to explore the phenomenon of seeking medical care among individuals in the community who had suffered panic attacks. This report presents the results of the screening of subjects for panic states, the first phase of the Panic Attack Care-Seeking Threshold study. Target numbers of subjects from 18 San Antonio census tracts were selected from 1980 census data so that the study population would be representative of

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the U.S. population in terms of age, gender, and race. The population of San Antonio is 54% Hispanic and 7% black (9). We chose census tracts—and the target number of subjects from each chosen census tract—to oversample black subjects. However, because of San Antonio's large Hispanic population, we did not attempt to achieve national representativeness with regard to ethnicity.

Following identification of a randomly selected starting point within each tract, clusters of three dwellings were surveyed at intervals of eight until the predetermined number of screenings were obtained. The adult to be screened in each dwelling was randomly selected by using the Kish method (10). If the individual was not available to be interviewed, two follow-up visits were attempted.

The screening consisted of a structured interview administered by two trained lay research assistants using the panic disorder portion of the SCID (7) and a demographic information sheet. Interviews were conducted in either English or Spanish at the subject's request. Spanish translations appropriate to the patient population were prepared by the research team. Even when subjects declined to be interviewed, the interviewers requested demographic information.

A limited-symptom attack was defined as a discrete episode of intense fear in a situation in which most people would not be afraid, associated with fewer than four of the 14 panic-related symptoms described in DSM-III-R. A panic attack was defined as a discrete episode of fear associated with at least four panic-related symptoms that developed within 10 minutes of onset of the attack. Panic disorder was defined as panic attacks—some of which were spontaneous and not due to an organic cause—occurring at least weekly for a 3-week period.

Subjects were classified as having limited-symptom attacks if they had experienced any limited-symptom attacks but had not experienced any full-blown panic attacks. Subjects were classified as having panic attacks if they had experienced any full-blown panic attacks but did not meet criteria for panic disorder. Subjects were classified as having panic disorder if they had at any time met the criteria for panic disorder.

The study was conducted from August 1989 through April 1990. A random sample of respondents were contacted by telephone to verify answers to a sample of interview questions. This procedure demonstrated that the data obtained were reliable.

Significance testing was performed with an alpha level of 0.05. Interval data were analyzed by using two-tailed t tests and analysis of variance (ANOVA) with Scheffé post hoc testing. Nominal or ordinal data were analyzed with chi-square, Wilcoxon, and Kruskal-Wallis tests. Differences in rates were assessed with z tests.

RESULTS

Of the 1,683 individuals contacted, 1,306 (77.6%) agreed to be interviewed. Refusal to participate was not

TABLE 1. Demographic Characteristics of 1,306 Subjects Participating in Study of Panic Disorder in San Antonio, Tex.

Characteristic	N	%
Gender		
Male	535	41.0
Female	771	59.0
Ethnicity/race		
Hispanic white	625	47.9
Non-Hispanic white	528	40.4
Black	126	9.6
Other	27	2.1
Annual income		
Less than \$10,000	581	44.5
More than \$10,000	582	44.6
Data missing	143	10.9
Educational level		
At least high school graduation	937	71.7
7–11 years	227	17.4
Less than 7 years	140	10.7
Data missing	2	0.2
Occupation		
Executives and professionals	57	4.4
Housewives	359	27.5
Retired and unemployed	279	21.4
Other	609	46.6
Data missing	2	0.2
Socioeconomic class		
I	34	2.6
II	112	8.6
III	210	16.1
IV	207	15.8
V	743	56.9
Language of structured interview		
English	1,194	91.4
Spanish	112	8.6

related to age, gender, educational level, annual income, socioeconomic status, or language preferred (English versus Spanish) but was related to ethnicity. Of the Hispanic white subjects, 12.2% refused screening, compared with 20.8% of the black subjects and 26.3% of the non-Hispanic white subjects ($\chi^2=45.18$, $df=2$, $p<0.001$).

The mean age of the subjects who agreed to be interviewed was 44.6 years ($SD=17.82$). Table 1 presents other demographic information for the respondents. There were more women than men in the sample, and Hispanic subjects constituted the largest ethnic group. The sample was skewed to the lower socioeconomic classes, and almost half of the respondents did not work outside the home. Only 8.6% of the subjects requested that the screening be conducted in Spanish.

Table 2 presents the crude lifetime prevalence rates for limited-symptom attacks, panic attacks, and panic disorder. Women had higher rates of panic attacks and panic disorder than men, but the difference between men and women was not statistically significant for limited-symptom attacks. No statistically significant differences in rates between Hispanic subjects and either non-Hispanic white or black subjects were found. Non-Hispanic white subjects had higher rates of limited-symptom attacks than did black subjects, and no black subjects had limited-symptom attacks. The combined rate of panic attacks and panic disorder for

TABLE 2. Crude Lifetime Prevalence Rates of Panic in 1,306 Subjects in San Antonio, Tex.

Item	Prevalence					
	Limited-Symptom Attacks		Panic Attacks		Panic Disorder	
	%	SE	%	SE	%	SE
Gender						
Men	1.3	0.5	3.2 ^a	0.8	1.5 ^b	0.5
Women	2.8	0.6	7.2	0.9	4.1	0.7
Ethnicity						
Hispanic	1.9	0.6	6.5	1.0	3.8	0.8
Non-Hispanic white	3.1	0.8	3.9	0.9	3.4	0.8
Black	0.0	—	8.0	2.4	5.5	2.0
Total	2.2	0.4	5.6	0.6	3.8	0.5

^a $z=3.02$, $p\leq 0.01$.^b $z=2.37$, $p\leq 0.05$.

black subjects (13.5%) was significantly greater than the combined rate in non-Hispanic white subjects (7.3%) ($z=2.23$, $p\leq 0.05$).

The presence of panic attacks was unrelated to age, occupational status, or socioeconomic status. Subjects with panic attacks had a higher mean income than those with limited-symptom attacks, who, in turn, had a higher mean income than subjects without attacks (Kruskal-Wallis $\chi^2=6.07$, $df=2$, $p=0.05$). Subjects with limited-symptom attacks had a higher mean level of education than those without attacks, but subjects without attacks had a higher mean level of education than those with full-blown panic attacks (Kruskal-Wallis $\chi^2=8.24$, $df=2$, $p=0.02$). Of the subjects with some panic symptoms, those with panic disorder did not differ demographically from those without the disorder except that they were younger ($t=2.43$, $df=56$, $p=0.02$).

DISCUSSION

Previous studies conducted on the general population have found lifetime prevalence rates of panic disorder of 1.2%–3.3% (11–17; unpublished 1986 paper by J.T. Marron et al.). Our finding of a 3.8% rate is higher than most other studies. The large ECA study (13, 15), which produced some of the lowest measured prevalences, used the DIS and applied DSM-III criteria. The use of the DIS may produce falsely low prevalences due to low sensitivity (6, 18) and/or low interrater agreement (18, 19). Most of the other studies reporting lower prevalences also used the DIS (12, 14). The frequency question of the DIS has low reliability, suggesting that DIS-measured prevalences may be falsely low (20).

Lifetime prevalence rates of 2.7% to 11.1% for panic attacks that do not meet criteria for panic disorder have been reported (11–13, 21, 22; unpublished 1986 paper by J.T. Marron et al.). The 5.6% prevalence of attacks not meeting criteria for panic disorder found in this study is thus in agreement with the literature.

The 2.2% prevalence of limited-symptom panic at-

tacks noted in this study is considerably lower than the rates of 4.8%–8.5% lifetime prevalence suggested in the literature (23, 24). However, to our knowledge ours is the first survey to specifically address limited-symptom attacks in a community sample.

The fact that panic attacks are more common in women is well accepted. In addition, the lack of a significant difference in prevalence between Hispanic and non-Hispanic white subjects is also in agreement with previous work (25, 26).

Although there has been no consistent association between race and panic in the literature, the ECA study (27) found a trend suggesting that the prevalence of panic in white subjects was lower than in nonwhite subjects. A study in Africa (28) found a higher prevalence of panic disorder than did the ECA study. In our current study we found a higher prevalence of any panic attacks—including panic disorder—in black subjects than in non-Hispanic white subjects but a lower prevalence of limited-symptom attacks. The prevalences in Hispanic subjects were between those of black subjects and non-Hispanic white subjects.

Perhaps the greatest importance of this study is in its estimation of the prevalence of limited-symptom attacks. There is evidence that the use of a cutoff of four panic-related symptoms to define a panic attack is somewhat arbitrary (1, 2, 29). Limited-symptom attacks may have consequences similar to those of full-blown attacks in terms of depression, suicide, and agoraphobia. If so, an understanding of their prevalence in the population is essential.

The limitations of this study include issues of sample representativeness and generalizability. Over 22% of the subjects contacted refused to be interviewed. Our results would be biased if the subjects who could not be contacted or refused to be interviewed differed from the respondents in their prevalence of panic states. Because San Antonio has relatively few black residents, census tracts with many black residents were relatively heavily sampled. Our results would be biased if these neighborhoods had other characteristics that influence the prevalence of panic.

The applicability of our findings in San Antonio, a city with a unique demographic composition, to the general population is also an issue. San Antonio's large Hispanic population may be different from other Hispanic groups in the United States (30); therefore, findings in this group of Hispanic subjects may not be generalizable to other groups. In addition, because our sample had relatively little education and low socioeconomic status, the applicability of these findings to groups with higher socioeconomic status could be questioned.

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Diagnostic and Substance Specificity of Carbon-Dioxide-Induced Panic

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***Objective:** The authors assessed the substance and diagnostic specificity of carbon-dioxide-induced panic since, in addition to the specific biochemical effects of inhaled carbon dioxide (CO₂), simple physiologic distress is also frequently implicated as a panicogenic factor during respiratory challenge studies with CO₂ in patients with anxiety disorders. **Method:** Eighteen patients with panic disorder, 20 with social phobia, and 23 psychiatrically normal subjects inhaled a mixture of 35% CO₂ and 65% O₂ for 30 seconds through a face mask. They also breathed for 30 seconds through a valve reducing the diameter of the airway. A double-blind, counterbalanced, randomized design was used. **Results:** In spite of important similarities between the two interventions, including the induction of equal amounts of subjective respiratory distress, carbon dioxide inhalation was significantly more potent than increased airway resistance in provoking panic in the anxiety disorder patients. The patients with panic disorder were significantly more sensitive to CO₂ than were the patients with social phobia or the normal subjects. **Conclusions:** Carbon dioxide inhalation appears to have a specific panicogenic effect in panic patients that goes beyond simple breathlessness.*

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While remarkably successful treatments for panic disorder are now available, the pathophysiology and etiology of the illness remain obscure. Biological challenges are among our most promising tools in clarifying these areas. Of the numerous agents capable of inducing panic in patients with panic disorder, carbon dioxide (CO₂) offers significant advantages. It is easily administered, well tolerated, and one of the most reliable panicogens.

Inhalation of various concentrations of CO₂ induces

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more anxiety and panic attacks in patients than in comparison subjects (1-4), suggesting that respiratory disturbance is a key component in the pathophysiology of panic. Several studies have showed biologically based CO₂ hypersensitivity among panic patients as determined by formal measurement of CO₂ sensitivity (5, 6, and unpublished 1987 study by D.B. Carr et al.). Although there have been negative results as well (3 and unpublished study of J. Zandbergen et al., 1991), these findings could reflect brainstem hypersensitivity to CO₂ (7).

On the other hand, breathing CO₂ induces, among other substantial physiologic and biochemical effects, increased ventilation, a subjective sense of dyspnea, exhaustion, and fatigue that the patient may associate with panic. An acute panic attack may thus occur through cognitive processes, such as misperception or misattribution (8-10). According to this view, specific biologic vulnerability to CO₂ is not involved and symptoms of physiologic distress, such as increased ventilation, dyspnea, and exhaustion, regardless of their origin, will be panicogenic.

In an earlier work (2) we compared the effects of CO₂ inhalation to room air hyperventilation. In spite of comparable exhaustion and fatigue, hyperventilation appeared significantly less powerful a panicogen than CO₂. With the design used in that study, however, the administration of CO₂ was not controlled by the sub-

ject, whereas hyperventilation was voluntary. It might be argued, therefore, that it was the lack of control over increased ventilation, rather than the specific biologic effect of CO₂, that produced those results. Furthermore, CO₂ inhalation always preceded room air hyperventilation, which may have introduced an order effect. We decided, therefore, to compare CO₂ inhalation to another form of involuntarily imposed increased ventilation and to counterbalance the order of interventions.

We theorized that reducing the diameter of the airway during room air ventilation would cause the patient to experience dyspnea and air hunger similar to that during CO₂ inhalation. Breathing against increased inspiratory resistance, however, does not lead to increase in arterial or brain CO₂ concentration (11, 12).

The goal of the present study was to compare the effects of inhalation against resistance to the effects of breathing CO₂ in patients with panic disorder, patients with social phobia, and psychiatrically normal subjects. Our hypothesis was that in spite of substantial distress in response to both conditions, the panic patients would perceive the inhalation of CO₂ as more anxiety and panic provoking. This would suggest a specific role for CO₂ in panic induction beyond symptoms of subjective physiologic distress, such as dyspnea.

METHOD

Subjects

Subjects were studied at three sites: an urban psychiatric hospital (site 1) and two suburban anxiety disorders clinics (sites 2 and 3). The staff was identical at all three sites; the procedure was identical at sites 2 and 3. The procedural differences at site 1 will be described.

We studied 18 patients (10 at site 1) with DSM-III-R panic disorder with agoraphobia, 20 patients with social phobia (all at site 1), and 23 psychiatrically normal subjects (18 at site 1). The ages of the three groups were as follows: panic disorder, mean=35.3 years (SD=9.6); social phobia, mean=34.3 years (SD=9.2); normal, mean=31.2 years (SD=8.6). The sex distributions were as follows: panic disorder, 12 women and six men; social phobia, four women and 16 men; normal, nine women and 14 men.

All subjects signed statements giving informed consent for the procedure after detailed explanation by the investigators. The subjects were all in good medical health and, except for benzodiazepines, had not taken medications for at least 2 weeks before the study. Benzodiazepines taken as needed were permitted up to 3 days before the study.

Procedure

On the morning of the experiment, after an overnight fast, the subjects arrived at the clinic, where the procedures were explained again. The consent form instructed the subjects that they would breathe CO₂ and/

or breathe against increased resistance. They did not know, however, whether they would receive two CO₂ challenges, two sessions of breathing against increased resistance, or one of each. They were not told about the timing or the order of the challenges. The doctor administering the procedure was kept blind to the challenges and to the diagnosis of the subject. The subjects were advised that they might experience anxiety or a panic attack but that the sensations were expected to be transient and not harmful in any way. The subjects were kept supine for the entire procedure.

Next the technician attached ECG leads, a blood pressure cuff, and, at site 1 only, a noninvasive respiratory monitor. The respiratory monitor continuously recorded respiratory rate and tidal volume; the instrument is described elsewhere (13). At site 1 only, a venous line was then started for periodic measuring of blood gases and pH.

At least 15 minutes after this apparatus was set up, an airtight mask was placed on the subject's face (time 0). The tubing of the mask extended to gas tanks outside of the room. The subject then breathed room air at his or her normal rate through the mask for 15 minutes. Baseline measures at 15 minutes included scores on the 28-item Acute Panic Inventory (14), a 10-point anxiety scale, a 10-point apprehension scale, and the 10-point Borg breathlessness scale (15).

At 20 minutes the subject received 30 seconds of either a mixture of 35% CO₂ and 65% oxygen (O₂) or increased inspiratory resistance; the order of these procedures was randomized and counterbalanced. The flow of the gas was set at 8 liters/min. The increased inspiratory resistance was achieved by inserting a commercially available valve into the airway, which reduced its diameter from 20 mm to 2 mm. Immediately after the intervention, room air breathing resumed and the rating scales were readministered. At 35 minutes a second set of baseline measures (Acute Panic Inventory and anxiety, apprehension, and Borg breathlessness scales) was obtained. At 40 minutes the subject received the other 30-second intervention (CO₂ or increased inspiratory resistance), and the same ratings were repeated.

A panic attack was defined as the development of at least four crescendo-type DSM-III-R panic symptoms in addition to an overwhelming sense of dread, fear of death or of losing control, and a desire to terminate the experiment or to flee. Physical symptoms alone did not suffice.

Data Analysis

Differences among the groups in sex distribution, rate of panic, order of interventions, and effect of order or site on response (panic or no panic) were tested with chi-square statistics. For the analysis of the baseline scores on the Acute Panic Inventory and the apprehension, anxiety, and breathlessness scales and of heart rate and blood pressure data, a 3×2 (Diagnostic Group by Sex) analysis of variance (ANOVA) was used. Tidal volume, respiratory rate, and minute ventilation were

averaged over the 5-minute preintervention period for each subject.

To assess the effect of the first intervention on the second baseline, the values at the two baselines (15 and 35 minutes) were also compared by means of ANOVA with repeated measures. A significant time effect would indicate a difference between the two baselines, suggesting an effect of the first intervention on the second baseline.

As a response analysis, differences between post- and preintervention values for each variable were first calculated for each subject and for both interventions. These were then used in a $3 \times 2 \times 2$ (Group by Sex by Intervention) repeated measures ANOVA. Order of intervention was added as a factor. For the respiratory measures the postintervention value was calculated as the average for the 30-second intervention period.

Significant ANOVA results were followed up by paired *t* tests. All significance levels are two-tailed.

RESULTS

There were no significant differences in mean age among the groups or between men and women: men, mean=33.7 years (SD=9.0); women, mean=32.9 years (SD=9.5). There was a significant difference in overall sex distribution among the groups ($\chi^2=8.6$, *df*=2, $p<0.01$). All ANOVAs consequently include sex as a factor. There were no significant Sex by Site interactions.

The proportions of subjects who received CO₂ first and those who received increased inspiratory resistance first were similar in the three diagnostic groups, and there were no Order by Sex interactions. The only site effect, a slightly higher mean blood pressure at sites 2 and 3, was probably due to the use of manual measurement at sites 2 and 3 and an automated method at site 1. Although the site 1 procedure differed from the other two, there were no statistically significant site differences for any other variable. We therefore decided to analyze the subjects as one group.

Panic Rate

The order of the interventions did not affect the rate of panic, and there were no site effects on the rate of panic in any group, indicating that the interventions, rather than the circumstances, determined the responses.

Thirteen of the 18 panic patients (72%), six of the 20 patients with social phobia (30%), and one of the 23 comparison subjects (4%) panicked in response to CO₂. Four panic patients (22%), one patient with social phobia (5%), and none of the comparison subjects (0%) panicked in response to increased inspiratory resistance. Except for one panic disorder patient who panicked only with increased inspiratory resistance, all the subjects who panicked in response to increased resistance panicked in response to CO₂ as well.

The rates of panic in response to CO₂ among the three groups were significantly different ($\chi^2=21.2$, *df*=2, $p<0.001$). Pairwise comparisons of CO₂ panic rates

showed a significantly higher rate in the panic patients than in either the patients with social phobia ($\chi^2=6.8$, *df*=1, $p<0.01$) or the normal subjects ($\chi^2=20.7$, *df*=1, $p<0.001$) and a significantly higher rate of CO₂ panic in the social phobia patients than in the normal subjects ($\chi^2=5.2$, *df*=1, $p<0.03$). The differences in rates of panic in response to increased inspiratory resistance did not reach significance.

Because of the low expected frequencies of panic in response to increased inspiratory resistance (fewer than five subjects in over 20% of the cells), we did not perform three-way chi-square tests contrasting the rates of panic with increased inspiratory resistance and CO₂. However, McNemar chi-square tests showed that CO₂ was significantly more potent than increased inspiratory resistance in provoking panic in patients with panic disorder ($\chi^2=7.4$, *df*=1, $p<0.01$) and patients with social phobia ($\chi^2=5.0$, *df*=1, $p<0.03$). The difference between the normal subjects' rates of panic with increased inspiratory resistance (0%) and CO₂ (4%) was not statistically significant.

Baseline Data

According to the 3×2 (Group by Sex) ANOVAs, there were significant differences among the diagnostic groups in several of the measures obtained before the first intervention (15 minutes): Acute Panic Inventory score, anxiety, apprehension, and breathlessness (Borg) scale scores, systolic blood pressure, pH, and PCO₂ (see table 1). The panic disorder and social phobia patients scored significantly higher on all four subjective scales at baseline and had lower pH than the normal subjects. While, unexpectedly, PCO₂ was somewhat higher in the panic disorder and social phobia patients than in the normal subjects, only the difference between the social phobia patients and the normal subjects was statistically significant ($t=-2.81$, *df*=33, $p<0.01$).

Significant sex effects were found for systolic blood pressure, PCO₂, and bicarbonate: the men had higher levels of each than did the women (see table 1). Because only one woman with social phobia completed the Borg scale, interpretation of the sex effect and the Sex by Group interaction was impossible for this variable.

There were no significant differences in heart rate, diastolic blood pressure, bicarbonate, respiratory rate, tidal volume, and minute ventilation among the three diagnostic groups (table 1). The only site effect was the previously mentioned higher baseline blood pressure at sites 2 and 3. The standard deviation for tidal volume was nearly significantly higher in the panic disorder patients (327.4 ml) than in the normal subjects (207.1 ml) ($F=2.50$, *df*=2, 39, $p<0.06$).

At the second baseline (before the second intervention), most of the significant and nonsignificant differences among the groups seen at the first baseline were maintained. The exceptions were PCO₂ (Diagnosis by Sex by Time: $F=5.67$, *df*=2, 37, $p<0.01$), bicarbonate (Diagnosis by Sex by Time: $F=5.78$, *df*=2, 36, $p<0.01$), and respiratory frequency (Diagnosis by Time: $F=3.41$,

TABLE 1. Baseline Psychological and Physiologic Measures for Male and Female Normal Subjects and Patients With Social Phobia or Panic Disorder Challenged With CO₂ and Increased Inspiratory Resistance

Variable	Normal Subjects		Social Phobia Patients		Panic Disorder Patients		ANOVA		
	M	F	M	F	M	F	Diagnosis	Sex	Interaction
Acute Panic Inventory score							F=5.94, df=2, 59, p<0.01	n.s.	n.s.
Mean	1.71	3.33	4.53	14.00	11.83	13.33			
SD	2.02	3.35	6.01	3.92	7.44	16.58			
N	14	9	15	4	6	12			
Anxiety scale score							F=6.75, df=2, 59, p<0.03	n.s.	n.s.
Mean	1.14	1.56	2.27	3.75	4.50	3.33			
SD	1.10	1.67	1.94	0.50	3.39	2.57			
N	14	9	15	4	6	12			
Apprehension scale score							F=12.40, df=2, 59, p<0.001	n.s.	F=4.76, df=2, 59, p<0.02
Mean	0.64	2.11	2.40	4.00	6.17	3.83			
SD	0.84	1.83	2.56	1.16	2.56	2.44			
N	14	9	15	4	6	12			
Borg breathlessness scale score							F=6.87, df=2, 36, p<0.01	— ^a	— ^a
Mean	0.41	0.33	0.50	4.00	5.50	1.20			
SD	0.63	0.37	0.58		4.36	1.82			
N	11	6	4	1	4	11			
Heart rate (beats/min)							n.s.	n.s.	n.s.
Mean	66.5	74.3	64.6	71.5	71.8	76.2			
SD	14.9	13.8	13.4	17.0	8.8	12.8			
N	13	9	16	4	6	11			
Systolic blood pressure (mm Hg)							F=6.39, df=2, 58, p<0.01	F=4.40, df=1, 58, p<0.05	n.s.
Mean	123.5	115.4	118.4	113.8	139.2	126.7			
SD	11.8	11.0	8.3	19.2	23.0	18.5			
N	13	9	16	4	6	11			
Diastolic blood pressure (mm Hg)							n.s.	n.s.	n.s.
Mean	71.2	69.6	69.3	64.5	71.0	70.1			
SD	11.7	10.1	9.3	12.0	9.3	13.4			
N	13	9	16	4	6	11			
pH							F=4.50, df=2, 44, p<0.02	n.s.	n.s.
Mean	7.38	7.37	7.37	7.35	7.35	7.36			
SD	0.02	0.02	0.02	0.02	0.01	0.02			
N	11	7	14	3	3	7			
PCO ₂ (mm Hg)							F=4.43, df=2, 44, p<0.02	F=6.63, df=1, 44, p<0.01	n.s.
Mean	46.7	43.4	49.1	48.3	51.3	46.4			
SD	4.3	1.6	3.9	2.6	2.9	2.3			
N	11	7	14	3	3	7			
Bicarbonate (meq/liter)							n.s.	F=16.35, df=1, 44, p<0.001	n.s.
Mean	27.2	24.9	27.6	25.9	27.9	25.7			
SD	1.5	1.2	1.5	1.8	1.9	1.6			
N	11	7	14	3	3	7			
Respiratory rate (respirations/min)							n.s.	n.s.	n.s.
Mean	16.7	16.4	16.1	21.4	16.4	18.0			
SD	3.7	3.7	3.1	5.2	2.0	3.7			
N	9	3	14	3	3	7			
Tidal volume (ml)							n.s.	n.s.	n.s.
Mean	671.3	597.4	814.0	659.9	857.5	676.9			
SD	259.5	134.6	150.6	382.8	482.5	267.9			
N	8	7	12	3	3	7			
Minute ventilation (liters/min)							n.s.	n.s.	n.s.
Mean	10.2	10.1	12.4	13.1	14.1	12.1			
SD	2.8	5.4	2.4	5.2	8.8	5.8			
N	8	7	12	3	3	7			

^aToo few female subjects in social phobia group for analysis.

$df=2, 39, p<0.04$). Again, these results require cautious interpretation given the small number of men who panicked and the small number of female patients with social phobia. There were no statistically significant differences in any variable between the first and second baselines. The absence of significant time effect or Group by Time interaction in the ANOVA for most variables indicates that the first intervention did not meaningfully affect the second baseline.

Response to Interventions

The $3 \times 2 \times 2$ (Group by Sex by Intervention) repeated measures ANOVAs of the differences between post- and preintervention values comparing the two interventions (see table 2) demonstrated that, compared to increased inspiratory resistance, CO_2 caused significantly greater increases in scores on the Acute Panic Inventory and the anxiety and apprehension scales, diastolic blood pressure, respiratory rate, tidal volume, and minute ventilation. As expected, CO_2 lowered pH across diagnostic groups, whereas increased inspiratory resistance had no significant impact on it. The intervention factor for the Borg score was not statistically significant ($F=2.54$).

The only diagnostic group differences were in respiratory rate and minute ventilation. In response to CO_2 the panic patients experienced significantly greater increases in respiratory rate ($t=-3.3, df=24, p<0.04$) and minute ventilation ($t=-2.8, df=23, p<0.02$) than did the normal subjects. The increases in respiratory rate ($t=-5.0, df=12.14, p<0.001$) and minute ventilation ($t=-2.7, df=23, p<0.02$) in response to CO_2 were also greater in the panic patients than in the patients with social phobia. As expected, significant sex effects were limited to tidal volume and minute ventilation.

Diagnosis by Sex by Intervention interactions were limited to systolic blood pressure and pH, and Diagnosis by Sex interactions were found for only PCO_2 and bicarbonate. The absence of meaningful sex effects and the number of obvious diagnostic differences that did not reach significance (see table 2) prompted a series of 3×2 (Group by Intervention) repeated measures ANOVAs using the same values. In this second set of ANOVAs, additional significant diagnostic differences emerged for Acute Panic Inventory score ($F=3.78, df=2, 56, p<0.03$) and anxiety score ($F=3.88, df=2, 56, p<0.03$). The panic disorder patients had significantly greater mean increases in panic score ($t=-3.76, df=25.43, p<0.002$) and anxiety score ($t=-2.52, df=38, p<0.02$) in response to CO_2 than did the normal subjects. To control for baseline differences, we performed a series of analyses of covariance and obtained essentially similar results. The only difference was a significant group effect for anxiety ($F=4.31, df=2, 52, p<0.02$).

DISCUSSION

As in our previous work, we have again shown that panic disorder patients are more susceptible to the pan-

cogenic effects of carbon dioxide than are normal subjects or patients with social phobia. In this experiment we have further shown that the mere induction of a subjective sense of dyspnea is insufficient to cause panic in panic disorder patients. While increased inspiratory resistance induced somewhat milder physiologic distress than CO_2 , it resulted in significant increases in most of the physiologic measures and in subjective anxiety. Therefore, increased inspiratory resistance appears to be a more convincing contrast than simple room air inhalation in confirming the substance specificity of CO_2 -induced panic. Although the subjects did not report any significant difference in the amount of breathlessness (Borg score) with CO_2 and increased inspiratory resistance, among the panic disorder patients the rate of panic in response to CO_2 was still significantly higher than the rate with increased inspiratory resistance. Thus, CO_2 must have a specific panicogenic effect that goes beyond simple breathlessness (16).

A number of interesting findings from this study deserve comment. The panic rate in response to 30 seconds of inhalation of 35% CO_2 and 65% O_2 was higher among the panic disorder patients (72%) than we previously found during double-breath inhalation of 35% CO_2 and 65% O_2 (50%) (17). The difference may relate to the greater control patients had over CO_2 administration with the double-breath procedure. With that procedure, the subject pressed a button on a mask to deliver CO_2 , whereas in the present study the CO_2 was delivered involuntarily through a face mask. Sander-son et al. (18) have shown the importance of the subject's sense of control in mediating the panic response to CO_2 inhalation among panic disorder patients.

Although the proportion of subjects who panicked in response to CO_2 was higher among the panic disorder patients than among the normal subjects or the patients with social phobia, the obvious diagnostic differences in Acute Panic Inventory score and anxiety level reached significance only when sex was not part of the analysis. This finding is probably due to the sex distribution in our study group (few male subjects who panicked and few female patients with social phobia). A significantly higher anxiety level in panic disorder patients than in comparison subjects has been consistently demonstrated during various panic-inducing laboratory procedures (19).

Although CO_2 inhalation did not produce significantly more breathlessness than did increased inspiratory resistance, it did produce significantly greater increases in diastolic blood pressure, respiratory rate, tidal volume, and minute ventilation, and it produced less drop in pH. It might be argued that these differences in physiologic stimulation could be responsible for the panic attacks. However, inspection of table 2 shows that the absolute differences between the interventions in diastolic blood pressure, pH, respiratory rate, tidal volume, and minute ventilation changes are very small. It is difficult to believe that this level of physiologic change is sufficient to provoke a panic attack.

TABLE 2. Change in Psychological and Physiologic Measures for Normal Subjects and Patients With Social Phobia or Panic Disorder Challenged With CO₂ and Increased Inspiratory Resistance

Variable	Increase After Intervention						Repeated Measures ANOVA			
	Normal Subjects		Social Phobia Patients		Panic Disorder Patients		Diagnosis	Sex	Intervention	Interaction
	CO ₂	Resistance	CO ₂	Resistance	CO ₂	Resistance				
Acute Panic Inventory score							n.s.	n.s.	F=27.04, df=1, 53, p<0.001	n.s.
Mean	5.91	1.46	13.53	3.63	18.72	4.69				
SD	7.20	4.92	16.12	6.78	12.90	16.85				
N	22	22	19	19	18	18				
Anxiety scale score							n.s.	n.s.	F=13.10, df=1, 53, p<0.002	n.s.
Mean	1.36	0.32	2.47	0.90	3.33	1.17				
SD	2.28	1.78	2.61	1.85	2.66	1.86				
N	22	22	19	18	18	18				
Apprehension scale score							n.s.	n.s.	F=14.64, df=1, 53, p<0.001	n.s.
Mean	1.27	0.05	1.68	0.47	2.83	0.50				
SD	2.12	1.33	2.61	1.35	2.73	1.58				
N	22	22	19	19	18	18				
Borg breathlessness scale score							n.s.	n.s.	n.s.	n.s.
Mean	1.27	0.82	2.20	0.80	3.46	1.41				
SD	1.49	2.17	3.19	1.92	3.72	2.09				
N	15	15	5	5	15	15				
Heart rate (beats/min)							n.s.	n.s.	n.s.	n.s.
Mean	6.25	2.05	5.30	2.60	6.24	2.88				
SD	10.48	11.83	6.50	5.49	21.04	4.83				
N	20	20	20	20	17	17				
Systolic blood pressure (mm Hg)							n.s.	n.s.	n.s.	F=4.80, df=2, 51, p<0.01 ^a
Mean	8.50	1.00	6.15	3.80	9.12	5.35				
SD	7.72	7.46	7.24	8.09	15.18	12.29				
N	20	20	20	20	17	17				
Diastolic blood pressure (mm Hg)							n.s.	n.s.	F=7.68, df=1, 51, p<0.008	n.s.
Mean	5.25	-1.20	4.50	-1.00	4.47	1.12				
SD	12.23	7.69	6.79	8.31	8.23	8.23				
N	20	20	20	20	17	17				
pH							n.s.	n.s.	F=22.21, df=1, 35, p<0.001	F=3.17, df=2, 35, p<0.05 ^a
Mean	-0.011	0.003	-0.003	0.002	-0.009	-0.004				
SD	0.015	0.006	0.007	0.011	0.006	0.005				
N	17	17	15	15	9	9				
PCO ₂ (mm Hg)							n.s.	n.s.	n.s.	F=5.18, df=2, 35, p<0.01 ^b
Mean	0.912	-0.150	-0.533	-0.213	0.600	0.660				
SD	2.718	2.109	2.371	3.412	2.909	1.315				
N	16	16	15	15	10	10				
Bicarbonate (meq/liter)							n.s.	n.s.	n.s.	F=4.23, df=2, 34, p<0.02 ^b
Mean	-0.194	0.081	-0.533	-0.033	-0.456	0.133				
SD	0.810	1.080	1.090	1.835	1.542	0.700				
N	16	16	15	15	9	9				
Respiratory rate (respirations/min)							F=5.47, df=2, 37, p<0.009	n.s.	F=4.81, df=1, 37, p<0.04	n.s.
Mean	2.9	1.5	0.9	1.3	7.9	3.5				
SD	3.5	3.1	2.2	2.8	4.1	4.0				

TABLE 2 (continued)

Variable	Increase After Intervention						Repeated Measures ANOVA			
	Normal Subjects		Social Phobia Patients		Panic Disorder Patients		Diagnosis	Sex	Intervention	Interaction
	CO ₂	Resistance	CO ₂	Resistance	CO ₂	Resistance				
Tidal volume (ml)							n.s.	F=10.55, df=1, 34, p<0.004	F=25.27, df=1, 34, p<0.001	n.s.
Mean	339.6	10.0	422.0	36.8	387.5	-61.5				
SD	384.8	321.6	436.1	197.7	410.9	119.2				
N	15	15	15	15	10	10				
Minute ventilation (liters/min)							F=8.71, df=2, 34, p<0.002	F=13.36, df=1, 34, p<0.002	F=43.64, df=1, 34, p<0.001	n.s.
Mean	7.5	0.3	7.4	1.4	13.8	1.6				
SD	5.7	3.7	6.0	3.4	5.5	4.1				
N	15	15	15	15	10	10				

^aDiagnosis by Sex by Intervention.^bDiagnosis by Sex.

Blood gas measures proved relatively unremarkable in this experiment. In our previous report on inhalation of 35% CO₂ (17) we also found that venous blood gases are not adequate to show changes during a very brief exposure to CO₂. Arterial blood is probably more informative in detecting subtle changes in acid-base status (20). This is the first time we, or to our knowledge any group, has ever shown panic disorder patients to have higher baseline PCO₂ levels than normal comparison subjects. We have no explanation for this statistically nonsignificant finding except that the number of panic disorder patients (N=10) for whom we measured blood gases was very small.

The nearly significant difference between the panic disorder patients and the normal subjects in the standard deviation of the baseline tidal volume, which was higher in the panic disorder patients, is consistent with our previous finding (13). Consistent with the hypothesis that panic disorder patients have a fundamental although subtle disturbance in respiratory physiology, this suggests a more chaotic respiratory pattern at baseline.

As in previous studies (13, 17), we found a rate of CO₂-induced panic among patients with social phobia that is midway between the rates for panic disorder patients and normal subjects. While patients with social phobia have been shown to demonstrate significant respiratory symptoms that closely correlate with their subjective anxiety (21), the meaning of this intermediate susceptibility requires further study. It appears, however, that at higher doses of CO₂ some diagnostic specificity is compromised although sensitivity may be increased. At the 5% CO₂ level we observed a 39% panic rate among panic disorder patients but none of our subjects with other anxiety disorders panicked (13), while at the 35% CO₂ level (current study) 72% of the panic disorder patients and 30% of the patients with social phobia had panic attacks.

We believe that the results of this study strongly suggest that the induction of subjective dyspnea is not the cause of CO₂-induced panic among patients with panic

disorder. These results do not, however, clearly tell us what aspect of CO₂ inhalation is responsible for panic. One possibility is that patients with panic disorder are biologically hypersensitive to the effects of CO₂ (7). We cannot entirely rule out the possibility, however, that other physiologic cues, such as increased blood pressure, are cognitive mediators of CO₂-induced panic. In attempting to determine the biological and cognitive factors that combine to cause CO₂-induced panic there is a need for further work using a variety of different control interventions.

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Bereavement After Homicide: A Synergism of Trauma and Loss

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Objective: The purpose of this study was to examine the intensity and relationship of trauma and responses to bereavement in family members after homicide. **Method:** The authors established an outpatient clinic that offered evaluation and supportive psychotherapy after the homicide of a family member. A standardized evaluation protocol was followed with 18 adults in order to detail variables of previous trauma, bereavement, and psychiatric disorder. Standardized measures of bereavement (Texas Revised Inventory of Grief) and trauma (Impact of Event Scale and Dissociative Experiences Scale) were also administered. **Results:** As a group, the 18 adults were characterized by a high frequency of antecedent psychiatric disorder (N=12), the homicide of a child (N=12), and an intensely idealized attachment to the deceased, whose image of violent dying recurred as a disorganizing flashback and dream. The measures of bereavement and trauma showed generally higher levels of intensity in the 18 subjects in the present study than in normal subjects and other cohorts of bereaved subjects. **Conclusions:** For those who have lost a family member through homicide, recognition of a relatively specific pattern of dysfunctional responses of grief and trauma would promote early identification and psychiatric referral.

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Abundant anecdotal material (1-4) and several clinical surveys (5-8) suggest that bereavement subsequent to homicide is specifically influenced by the mode of dying and that the indirect exposure to homicide creates acute, and sometime chronic, signs and symptoms of posttraumatic stress phenomena. The intense traumatic response of numbing and intrusion coexists with the responses to loss of longing and sadness. This coexistence of trauma and loss is presumed to create a synergism of delayed recovery. The therapeutic inference drawn from clinical studies is that treatment of posttraumatic stress phenomena specifically associated with homicide takes precedence over treatment of the grief associated with the death. There is a strong therapeutic presumption that "grief work" must await recovery of a more stable psychological autonomy, which was undermined by the overwhelming trauma of homicide.

Validation of these preliminary findings must await systematic surveys differentiating bereavement after homicide from other forms of bereavement. Prospective and controlled studies to document the specific affects

of bereavement after homicide are stymied by the resistance of potential research subjects (a minority will volunteer for study) and the relative absence of specific and reliable measurements.

THE STUDY

The Support Project for Unnatural Dying was initiated in 1990 at a large county hospital in Seattle. Serving as the major trauma center for the greater Seattle area, this institution also houses the county medical examiner's office. These two resources are in daily contact with family members of those who die from unnatural causes.

Our clinical purpose was to offer early assessment and preventive intervention if indicated, since prospective study has documented the effectiveness of short-term psychotherapy in preventing complications from bereavement after a traumatic death (9). A prospective research survey, using serial standardized measures, was employed to document levels and patterns of dysfunction and their change over time. We hypothesized that measures of grief, trauma, and dissociation would be abnormally high and would support anecdotal and clinical reports that have long suggested a pattern of dysfunctional grief and posttraumatic stress phenomena after homicidal death. While the project also served bereaved families after suicide and accidental deaths, this preliminary report will review our first year's work with bereavement after homicide.

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Subjects

During the early phase of our project, we appreciated that we were attracting a highly dysfunctional cohort of young adult family members. The vast majority of subjects were referred by support groups or therapists because their efforts to assist had been ineffective. Few family members (less than 20%) called the project for primary evaluation and assistance despite our efforts, through the media and the medical examiner's office, to announce our willingness to assist at no charge. It must be emphasized that these subjects represent a biased group whose response to a homicide was so intense and dysfunctional that they or their therapists sought more specialized consultation. We recognize that these subjects do not, in all probability, represent a normative response to homicide.

Procedure

Data were gathered from 18 adults referred for consultation. A questionnaire was developed to gather demographic data as well as information detailing variables of previous psychiatric adjustment, the homicide, relationship with the deceased, and sequelae of the trauma. Standardized measures of grief (Texas Revised Inventory of Grief) and psychological trauma (Impact of Event Scale and Dissociative Experiences Scale) were included in the evaluation protocol. These measurements were chosen because of their demonstrated sensitivity and reliability in measuring dysfunctional responses in studies of nonpatients and patients, thus allowing a comparison of scores with our cohort. In brief, these tests are self-administered symptom checklists that quantify the intensity of past and present symptoms of grief (Texas inventory) and event-related symptoms of traumatic intrusion (flashbacks and dreams of the trauma) and traumatic avoidance (compensatory withdrawal from associative stimuli of the trauma) (Impact of Event Scale). Dissociation (the psychophysiological process that diminishes the conscious registration of overwhelmingly fearful experience) is commonly cited as a compensatory response to trauma. The Dissociative Experiences Scale was included to measure symptoms of dissociation in our cohort.

RESULTS

Subjects

The 18 subjects were predominantly young adults (mean age=39 years, range=22–53), white (15 were white, two black, one was Latino), and women (14 women, four men); most were unmarried (eight were married, five single, three divorced, one was separated, one widowed). Their mean annual income was \$30,000 before the death, and it fell to \$16,000 the year after the death. The subjects were well educated (mean years of education=14) and predominantly nonreligious (N=10).

Nearly all of the subjects (N=16) had experienced the

death of a family member from natural causes in the past, and this had not required clinical support or consultation. A remarkably high proportion (N=12) had a previous history of psychiatric consultation; the majority had had brief outpatient therapy (11 had had psychotherapy, six group psychotherapy, five pharmacotherapy); three subjects had required hospitalization. Past psychiatric diagnoses included major depressive disorder (N=5), anxiety disorder (N=7), and chemical dependency (N=8). Subjects were questioned in depth about past history of trauma. It was reported by a minority of subjects (five reported childhood physical abuse and two childhood sexual abuse); however, one-half of the subjects considered their family of origin to be dysfunctional.

Homicide and Traumatic Sequelae

A mean interval of 2.5 years (range=1–180 months) had elapsed since the homicide. The young age of the victims (mean age=28 years, range=15–63) reflected the preponderance of deaths of children (12 children, two siblings, two spouses, two parents, and two acquaintances). Two of the homicides involved multiple deaths (husband and daughter, and two acquaintances), and three of the homicides were committed by members of the same family. Shooting was the most frequent means of homicide (N=10), and intoxication with drugs or alcohol was a common association (N=8).

As a group, the subjects did not feel that they received social support (10 felt supported by friends, six by family, six by co-workers, and four by their church). Their perception of the deceased remained highly idealized and vividly persistent.

While only one of the subjects had actually witnessed the homicide, all but one experienced an intense and terrifying identificatory reenactment of the events of the murder. The scenario contained fragments from police and media reports as a substrata, but abundant and elaborate fantasized projections embellished the visual imagery. These images of reenactment recurred multiple times during the day as intrusive images that interfered with concentration and activity. This traumatic imagery became a dreaded epiphenomenon for these subjects, serving as a recurring source of anxiety and sense of impotence. The same image of reenactment emerged as a recurring nightmare that interfered with sleep in the majority of subjects (N=13). Images of rescue, revenge, and reunion were rarely reported as flashbacks or dreams.

The subjects were offered participation in a weekly support group. In addition, most of the subjects (N=12) were treated with medications to modulate anxiety (anxiolytics, N=10), insomnia (hypnotics, N=6), and depression (antidepressants, N=4).

Grief and Trauma

Eighteen subjects completed the Impact of Event Scale and the Texas Revised Inventory of Grief, and 16

TABLE 1. Scores on Impact of Event Scale Reported in Selected Studies

Study	Trauma	Subjects		Time Since Death (years)	Mean Score		
		Cohort	N		Intrusion	Avoidance	Total
Horowitz et al. (10)	None	Normal subjects	110		6.1	6.6	12.7
Zilberg et al. (11)	Natural death	Nonpatients	37	0.5	13.5	9.4	22.9
	Natural death	Psychiatric patients	35	0.5	21.2	20.8	42.0
Amick-McMullan et al. (6)	Homicide of relative	Nonpatients	16	2.5	24.6	16.9	41.3
Present study	Homicide of relative	Psychiatric patients	18	2.5	28.7	19.8	48.9

TABLE 2. Scores on Texas Revised Inventory of Grief Reported in Two Studies

Study	Trauma	Subjects		Time Since Death (years)	Score			
					Part 1 (past feelings)		Part 2 (present feelings)	
		Cohort	N		Mean	SD	Mean	SD
Zisook (12)	Natural death	Widows (nonpatients)	152 (part 1) 143 (part 2)	3.0	17.8	—	37.8	—
Present study	Homicide of relative	Psychiatric patients	18	2.5	29.7	6.9	54.8	10.7

subjects completed the Dissociative Experiences Scale. With this small group, the significance of the data was necessarily limited. Differential analysis of the data showed no significant association between grief and trauma and either gender or the interval of time since the homicide.

The mean scores on each of the three measures were generally higher than those reported by other studies (tables 1–3). The significance of the comparatively higher scores is weakened by multiple, unmatched variables between the groups studied. However, the markedly high mean scores of the subjects in the present study reflect the extremity of their grief (Texas Revised Inventory of Grief) admixed with severe trauma (Impact of Event Scale, Dissociative Experiences Scale).

DISCUSSION

The findings from these subjects who presented for psychiatric evaluation and intervention will be of potential relevance to clinicians working with family members after a homicide. Recognition of a relatively specific pattern of dysfunctional responses of grief and trauma would promote early identification and psychiatric referral. The combination of intense grief (idealized pining, longing, and guilt) and intense traumatic imagery (involuntary flashbacks and dreams of reenactment, although the murder was rarely witnessed) should alert the clinician to the potential for a dysfunctional response.

The piercing and recurring reenactment imagery appeared to complicate recovery. The subjects reported that this imagery had independent effects in creating anticipatory dread and fear of its involuntary repetition. Active efforts to avoid any cues that corresponded to homicidal imagery (such as television news reports or shows depicting violence, geographic areas associated

with the homicide, or viewing of guns or other means of homicide) were a common response. The psychological basis for involuntary processing of intrusive traumatic imagery and compensatory avoidance remains obscure (15, 16). The nearly unanimous reporting of traumatic imagery occurring on a daily or weekly basis for months and years differentiates this group from nonpatients, a minority of whom present with such frequent or enduring imagery (6). Presumably the antecedent psychiatric morbidity, the intense and idealized relationship with the deceased (particularly so with a child), and the high utilization of dissociation compromise the neuropsychological accommodation of the traumatic experience. These repetitive aversive images are so involuntary and stereotypic that they might conform to the neurological model of kindling (17). Perhaps the intense repetition of the fantasized death scene promotes a neurogenic "locus" of the percept. Once voluntarily or spontaneously activated, the cascade of images and affects proceeds in an unalterable procession and rush.

We and other clinicians have recognized the primacy of PTSD phenomena over grief phenomena in nonrecovery (18–21). Disintegratory effects of traumatic imagery and avoidance on cognition, affect, and behavior impair the more introspective and reflective demands of acknowledging and adjusting to the loss. While acknowledgment of the loss is a fundamental theme in therapy with this group of intensely traumatized subjects, the initial goal of treatment includes moderation of the intrusive/avoidance response.

Psychotherapeutic techniques to moderate traumatic imagery and avoidance focus on recovery of psychological autonomy—the highly subjective capacity to maintain a relative calm and transcendence while immersed in chaotic and disintegrating imagery. Paradoxically, the initial therapeutic objective includes recovering the subjective state of autonomy that was lost

TABLE 3. Scores on Dissociative Experiences Scale Reported in Selected Studies

Study	Subjects		Mean Score
	Cohort	N	
Bernstein et al. (13)	Normal subjects	34	5.7
Bernstein et al. (13)	Agoraphobic patients	29	7.9
Present study	Bereaved subjects	16	20.0 ^a
Bernstein et al. (13)	Schizophrenic patients	20	21.3
Carlson et al. (14)	Cambodian refugees	15	37.1
Bernstein et al. (13)	Patients with multiple personality disorder	20	56.3

^aSD=11.99.

by the deceased during the terminal moments of his or her life. Indeed, a common finding with traumatic reenactment imagery is the strong belief that the victim's last thoughts were overwhelmed by terror and helplessness. To some extent, then, this loss of autonomy may be viewed as an identificatory phenomenon. It would also seem probable that these subjects' antecedent psychological vulnerabilities would diminish their capacity for autonomy. Therapy begins by reinforcing skills and activities that allow calming and transcendence (guided imagery, relaxation techniques, recovery of positive images of the deceased). At the same time, the reenactment imagery becomes a shared experience with the therapist, and together the patient and therapist will examine the dying (not only through words but with the patient's drawing of the imagery) in order to reconstruct or restore a mutually enacted survival of the dying experience.

Group therapy offers a rich format for the exchange and mutual survival of death imagery. The subjects were offered a weekly support group, and the empathic awareness and efforts to help others master death imagery have been supportive. Pharmacotherapy has also been indicated. The majority of the subjects (N=12) were treated with medications to modulate anxiety, insomnia, and depression.

This study does not offer sufficient rigor or control to validate the effectiveness of short-term intervention. Considering the psychological vulnerabilities of this cohort before the traumatic aftermath of the homicide, assumptions of improvement are necessarily limited. Instead, we see our project as a resource of ongoing support in the long-term effort to adapt.

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Sexually Assaultive Male Juveniles: A Follow-Up

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***Objective:** The purpose of this study was to investigate the adult outcome of a group of male juveniles who committed sexual assault. **Method:** Nineteen sexually assaultive male juveniles and a comparison group of 58 violent juveniles were studied over an 8-year period through use of criminal records and clinical interviews. **Results:** Although in adolescence the two groups were similarly violent, on follow-up those who had committed sexual assault were significantly more likely to commit adult sexual offenses. They also committed significantly more violent nonsexual offenses. Childhood sexual abuse, especially by females, was associated with adult sexual offenses. **Conclusions:** Sexually assaultive delinquents are at particularly high risk for subsequent violence. Hence, special efforts must be made to treat these delinquents in adolescence. Prevention of violent sexual behavior must include improved methods of detecting sexual abuse, especially that perpetrated by older females.*

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The purpose of this paper is to report the adult criminal outcome and early sexual experiences of a group of sexually assaultive male juvenile delinquents. In a previous study reported in this journal (1), we compared the neuropsychiatric status and delinquent behavior of a group of 17 sexually assaultive male juveniles with that of a group of 61 violent, incarcerated male adolescents who did not commit sexually assaultive acts. The adolescent sex offenders were found to be similar to the violent comparison group both in their neuropsychiatric status and in the severity of their nonsexual violent behavior. That is, similar proportions in both groups manifested psychotic symptoms such as paranoid ideation (73% versus 83%) and auditory hallucinations (47% versus 41%). Similar proportions had signs of major neurological problems, such as a history of seizure disorders or an abnormal EEG (24% versus 31%), and scores on intelligence tests for both groups were in the low normal range. A history of physical abuse was also equally prevalent (76% versus 76%). Similar proportions of sexually assaultive juveniles and juveniles in the violent comparison group had been physically abused by their mothers (46% versus 43%) and fathers (58% versus 58%).

This paper describes what happened to these two ostensibly similar groups of adolescent boys over a period of approximately a decade between discharge from juvenile corrections and follow-up. It also sheds light on certain early experiential differences between the groups that were not identified when they were evaluated as adolescents.

Prospective studies of young sex offenders studied through adolescence report a recidivism rate of 2%–14%. For example, in a 6-year follow-up study of 108 male adolescent sex offenders, Doshay (2) found that only two subjects committed another offense. Atcheson and Williams (3) reported a 3% recidivism rate for male adolescents. In a more recent report, Smith and Monastersky (4) studied a group of 112 juvenile sex offenders for a mean period of 17 months. Fourteen percent committed a sexual offense during the follow-up period.

To the best of our knowledge there have been no reports in which a group of sexually assaultive male adolescents has been studied into adulthood. Thus, little is really known about the long-term criminal outcome of adolescent sex offenders. We therefore welcomed the opportunity to investigate the adult outcome of our group of sexually assaultive juveniles.

METHOD

Subjects

The present study group consisted of 19 sexually assaultive male juveniles and a comparison group of 58 boys who had committed violent but nonsexual acts. Both groups of boys were incarcerated in the only cor-

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rectional school in Connecticut in the late 1970s. In the original study (1) there were 17 sexually assaultive male juveniles and 61 violent comparison subjects. Information obtained subsequent to publication of the original study revealed that two comparison subjects had also committed sexually assaultive acts as juveniles. Therefore, in the present study, they were transferred from the comparison group to the sexually assaultive group. An additional comparison subject died shortly after discharge from juvenile corrections and was dropped from the follow-up study. Thus, the violent comparison group was reduced to 58 subjects, and the sexually assaultive group increased to 19.

Of the 19 sexually assaultive juveniles, four were white, 11 were black, three were Hispanic, and one was Native American. Of the 58 comparison subjects, 19 were white, 26 were black, and 13 were Hispanic. Subjects' ages averaged 15 years at the time of the initial evaluation. At the time of the clinical follow-up study the mean age was just over 24 years. The majority of subjects were from socioeconomic classes IV and V according to the Hollingshead and Redlich criteria (5).

Follow-Up

Adult criminality. Eight years after the original study and with proper assurances of confidentiality, follow-up information regarding adult criminality was obtained from the Federal Bureau of Investigation, the Connecticut State Police, and the Connecticut Department of Corrections. The following data were recorded: 1) the number, nature, and timing of arrests, and 2) the duration and timing of incarcerations.

Clinical data. Extensive efforts were made to contact all subjects and conduct personal interviews. It took over 4 years and, on average, 11 attempts per subject to locate and interview subjects and close friends and relatives. Thus, the entire study spanned 12 years. The follow-up interview took place, on average, approximately 9 years after the adolescent's original evaluation. The follow-up interview covered experiences subsequent to discharge from juvenile corrections, including education, job training, employment, interpersonal relationships, and medical and psychiatric treatment. In addition, subjects were questioned regarding histories of physical and sexual abuse.

After informed consent was obtained, follow-up interview data were gathered on 17 of the 19 sexually assaultive juveniles and 41 of the 58 comparison subjects. In four cases, when subjects were unavailable, close relatives agreed to be interviewed instead. Because the attitudes and openness of subjects varied (and in four cases the informant was not the subject but a close relative), the completeness of follow-up data also varied. Thus, the group size (i.e., the number of subjects) for different variables is not always the same.

Sexual abuse. At the time of the original study we were less sophisticated regarding the importance of gathering data on sexual abuse. At the time, all available records were reviewed to assess histories of sexual

abuse, but these issues were not explored systematically and in depth in all clinical interviews. In contrast, issues regarding sexual abuse were systematically explored in all follow-up interviews.

Subjects were considered sexually abused if either their early records or follow-up interviews indicated that they had been directly involved with an adult or older child for purposes of the adult's or older child's sexual gratification. Witnessing the sexual activities of others was not categorized as abuse. Activities that constituted abuse included being forced to stimulate the adult or older child manually or orally, being manually or orally stimulated by the adult or older child, being forced to perform intercourse, or being sodomized with objects or sexual organs. Because of the unique nature of sexually abusive experiences, arbitrary age differences between victim and perpetrator were not used; however, in this study, the perpetrator was never less than 5 years older than the victim. Victims did not have to regard the sexual experience as abusive as long as their experiences were of the nature described previously.

FINDINGS

Adult Criminal Outcome

By age 24 years, seven (37%) of the 19 sexually assaultive juveniles had an adult criminal record of one or more first- or second-degree sexual assaults subsequent to discharge from juvenile corrections. Unfortunately, criminal records did not specify the age or sex of the victims. Only six (10%) of the violent comparison group had a record of adult sexual assaults; this difference was significant ($p=0.01$, Fisher's exact test). Noteworthy was the finding that only subjects who had been identified as sexually assaultive juveniles committed multiple sexual offenses in adulthood. For example, one subject committed nine adult sexual offenses, although he was at liberty for only 9.6 months of the follow-up period. Another subject committed five adult sexual offenses in a period of 10.8 months.

Sexually assaultive juveniles did not limit their adult violence to sexual crimes. Seventeen (89%) of the 19 were arrested as adults for other kinds of violent offenses (i.e., murder, kidnapping, robbery, and assault), compared with 40 (69%) of the 58 in the comparison group ($p=0.08$, Fisher's exact test). What is more, when corrected for years at liberty, the total number of violent offenses committed per year by the sexually assaultive juveniles was more than twice that of the comparison group (4.9 versus 2.2; $t=-2.05$, $df=74$, $p=0.04$). Thus, as adults, sexually assaultive juveniles were significantly more dangerous than other violent juveniles.

Other follow-up studies of sexually assaultive adolescents have reported lower rates of recidivism. However, those studies terminated after a relatively brief period of time (e.g., 17 months). As noted, Smith and Monastersky (4) reported a 17-month recidivism rate of 14%. We wondered whether, had we ended our follow-up

after 17 months, our findings would have been similar to those of Smith and Monastersky. We therefore calculated the rate of recidivism for our subjects over a 17-month period after discharge from juvenile corrections. The resulting recidivism rate in our group was 21%. This rate is higher than that of Smith and Monastersky but just over half the rate of 37% calculated for the 8-year follow-up period.

Subjects' Candor Regarding Sexual Violence

How candid were subjects regarding their own sexual assaultiveness? By definition, all sexually assaultive juveniles had been sexually assaultive during adolescence. However, at the time of follow-up, interviewers were blind to subjects' prior sexual or aggressive behavior. When they asked the sexually assaultive juveniles whether they had ever forced sex upon someone else, only two (12%) of the 17 who were interviewed admitted to such behavior. In the comparison group, three (7%) of the 41 subjects interviewed admitted to having forced sex upon someone else. (Only two of the three were arrested for it.)

Early Sexual Experiences

Were early sexual experiences associated with the nature of later criminal behavior? Data regarding early sexual experiences were incomplete. Only 15 of the sexually assaultive juveniles and 35 of the violent comparison group were even willing to discuss their first sexual experience. However, on the basis of information gathered from those subjects who were willing to talk with us, we found that, on average, the sexually assaultive juveniles had sexual intercourse at a significantly earlier age than the comparison group (11.9 years versus 13.6 years; $t=2.41$, $df=48$, $p=0.02$). At the time of the study, this age was well below the average age for first intercourse among adolescent males in the United States. According to a study performed by the National Center for Health Statistics (6) between 1982 and 1988, a period shortly after the present study, the average age at which males experienced their first sexual intercourse was 15.7 years.

Histories of Sexual Abuse

Of the 17 sexually assaultive juveniles for whom clinical follow-up information could be gathered, seven (41%) either reported or had documented histories of having been sexually abused in childhood. Only one subject was abused by a male, his stepfather. This man's behavior was also known to the subject's mother. The subject's age at the time of the abuse and its duration were not documented. The other six subjects were abused by females. Two were abused during their early grade school years by teen-age baby sitters. One of these, at age 8, was made to have intercourse with his sitter. Another subject, beginning at age 5, was fondled by and was forced in turn to fondle his sister and her

girlfriend. This behavior was thought to span a period of several years. Three other subjects were molested by older women who lived in their neighborhoods. One of these women was a friend of the subject's mother. The average age at which sexual victimization by females occurred was 9.4 years (median=9.3).

Of the 41 subjects in the violent comparison group who were interviewed, nine (22%) had histories of having been sexually abused in childhood: three by males, five by females, and one by both a male and a female. In this group two boys, in adolescence, were involved with an older male whom they subsequently murdered; another boy was molested by an uncle at age 9 years. Two of the comparison subjects were victimized by women in their neighborhoods, one boy at age 12 years, the other at age 13. One subject, at age 7, was sexually abused by a family friend who baby-sat for him. This abuse lasted for approximately 1 year. Another subject reported an ongoing sexual relationship during his 14th year with his junior high school art teacher. Still another had an ongoing sexual relationship starting at age 12 with his mother's girlfriend; this relationship eventually included the boy's two younger brothers as well. The boy who was victimized by both a male and a female had been sodomized by a 15-year-old when he was 10 years old. Subsequently, when he was 15, he became involved in an ongoing sexual relationship with a 37-year-old woman. In this group, the average age at which sexual victimization was perpetrated by a male was 12.3 years (median=12.5); the average age at which victimization was perpetrated by a female was 12.2 years (median=12.5). Thus, subjects in the sexually assaultive group were sexually abused at an earlier age than subjects in the comparison group; however, this difference did not reach conventional levels of significance.

It should be highlighted that of the 16 sexually abused subjects in the two groups, 12 (75%) were victimized by females. This finding contrasts with current statistics which indicate that the majority of perpetrators of child sexual abuse are male (7-9). Of special note, all of the sexually assaultive juveniles who committed multiple adult sexual offenses had early documentation of or admitted on follow-up to having been sexually abused as children.

DISCUSSION

In our previous paper we compared juvenile sex offenders to a violent, nonsexually assaultive group of delinquents and found the two groups to be similar in their violent nonsexual behavior. After following these two groups into adulthood, we found that they were no longer similar. In adulthood the juvenile sex offenders were far more violent, committing substantially more sexual and violent nonsexual offenses. What is more, those with evidence of having been sexually abused as children became repetitive adult sex offenders.

Although we could obtain histories of sexual abuse in only 41% of the sexually assaultive juveniles, we had

reason to believe that the actual proportion was much higher. For example, several subjects, who in adolescence denied having been sexually abused, admitted on follow-up that sexual abuse had occurred. Others in adulthood gave histories of having had sexual intercourse as children with individuals who were many years their senior, yet they minimized the abusive nature of these encounters. For example, one of the sexually assaultive juveniles, who had sexual intercourse with his teen-age baby sitter when he was only in first grade, described the encounter as normal, explaining, "It happens to everyone."

Still other subjects, some of whom were known to have been the most severely sexually abused, had clouded memories or no recollections at all of their abuse. For instance, one sexually assaultive juvenile, who had been chronically sexually abused by his older sister and her friend, was hospitalized during adolescence after a thermometer was inserted through his penis into his bladder. His impaired recollection of the event made it impossible to determine the perpetrator of the act even though it resulted in a 6-week hospitalization. Our subsequent clinical work with sex offenders has convinced us of the likelihood that other subjects in the present study used dissociative mechanisms to keep intolerable physically and sexually abusive experiences from consciousness. This kind of defense mechanism would prevent us from obtaining a full appreciation of the scope of sexual abuse in this population.

Most surprising was the finding that contrary to the literature, which states that the overwhelming majority of sexual abusers are men (7-9), 75% of the perpetrators of sexual abuse upon our subjects were females. One explanation for this discrepancy with the literature may be the fact that most of the literature regarding sexual abuse of boys deals with general populations of abused boys, whereas the two groups in the present study consisted of offenders.

Another explanation for our finding of predominantly female abusers may be the ways in which questions regarding sexual abuse were asked of the subjects. Many subjects did not perceive having sexual relations with older women during childhood as abusive. Thus,

in response to questions such as, "Did anyone ever bother you sexually?," they would say "No." It was, rather, in response to such questions as, "How old were you and your partner when you first had intercourse?" and, "Have you ever had sexual relations with someone much older?," that many subjects reported significant age differences between their partners and themselves that were great enough to be considered sexual abuse (e.g., 17 versus 9 years of age). These kinds of questions enabled us to uncover instances of sexual abuse that would otherwise not have been detected.

If our findings are representative of the larger population of sexually assaultive juveniles, then we must conclude that these juveniles are an extraordinarily violent group who continue to commit sexually assaultive offenses, as well as other violent nonsexual offenses, into adulthood. If we are to begin to prevent this violent sexual behavior, we must improve our methods of detecting sexual abuse. We must also recognize that although sexually assaultive juveniles often deny having been victimized, they have frequently been victims of sexual abuse, and this abuse has often been by females.

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Partial Hospitalization (Day Treatment) for Psychiatrically Ill Elderly Patients

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Objective: The purpose of this paper is to present initial findings from a retrospective chart review of geriatric day treatment patients in order to focus attention on this potentially important area, add to the limited database in this area, and generate hypotheses for future investigations. **Method:** Data were abstracted from the charts of 100 geriatric day treatment patients over a period of approximately 5 years (1985–1989). Descriptive, univariate, and multiple regression techniques were used to describe the patients and identify variables associated with their outcomes. **Results:** The typical patient in this program was a widowed white woman in her 70s who suffered from a depressive disorder. During the initial treatment period (usually approximately 3 months), 57% of the patients experienced some clinical improvement. Variables associated with a favorable outcome included diagnosis of a mood disorder rather than a psychotic disorder, better initial functional status, greater initial social support, fewer stressful events during treatment, and longer duration of treatment. **Conclusions:** Geriatric day treatment can be effective and merits further study as a mode of treatment for psychiatrically ill elderly patients.

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Partial hospitalization is a program of intensive, coordinated, and structured psychiatric services in an ambulatory setting (1); day treatment is on the less intensive end of the spectrum of partial hospitalization services. Although this form of service delivery has primarily been used by young and general adult clinical populations, a confluence of trends in health care (e.g., efforts at cost containment, health services research) makes partial hospitalization particularly attractive as a potential form of mental health service delivery for the elderly. Although there has been a proliferation of partial hospital programs in the private sector recently (2), there is a paucity of empirically derived information on which to base guidelines for clinical services.

The purpose of this paper is to present initial findings from a retrospective chart review of geriatric day treatment patients in order to focus attention on this poten-

tially important area, add to the limited database in this area, and generate hypotheses for future investigations. More specific purposes are to characterize geriatric day treatment patients, to identify their outcomes, and to explore variables of interest that are associated with outcome.

BACKGROUND

Published studies of partial hospitalization have generally focused on young adults with chronic mental illnesses such as schizophrenia. Controlled studies have compared the efficacy of partial hospitalization with that of inpatient (3–22; unpublished 1977 paper by Hirsch) and outpatient (23–28) services and have addressed cost effectiveness as well (9, 11, 20, 23, 29–34). In general, these studies found that partial hospitalization compares favorably with more traditional modes of treatment in terms of both clinical outcomes and costs (35–37), although it has been noted that methodological limitations preclude definitive conclusions until more rigorous empirical studies are performed (4, 38, 39).

None of the studies cited here addressed either the efficacy or the costs of partial hospitalization for geriatric patients. In general, there is a paucity of information on psychogeriatric partial hospitalization. Most of the literature on geriatric day services focuses on day care (40, 41), which emphasizes social and/or custodial services rather than provision of specific treatments.

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A review of the few studies pertaining to day treatment/partial hospitalization for elderly patients suggests that this modality may be helpful for some patients, but no randomized studies have been reported. Bergener et al. (42) compared the 4-year course of 206 inpatients with that of 75 patients who had received only partial hospital treatment during the first 2 years. More of the partial hospitalization patients than inpatients were released to their homes, but readmission rates of the two groups were not significantly different. Eskew et al. (43) conducted a retrospective chart review of 93 patients in a day treatment program and found that 55.6% were rated as improved between admission and follow-up evaluation, 33.3% were unchanged, and 11.1% were worse. Patients with affective disorders fared better than did those with organic brain disorders. Wagner (44), in a description of a partial hospitalization program in a free-standing psychiatric facility in Ohio, noted that outcomes appeared to be favorable, but empirical outcome findings have not yet become available. Other reports have been limited to program descriptions (45–53).

HYPOTHESES

Based on the literature, our knowledge of our patient population, and our own clinical experience, we formed two tentative hypotheses for this study. The first was that most elderly patients would experience some improvement in symptoms and daily functioning and that a minority would get worse and not be able to complete the program. The second was that the following variables would predict a favorable clinical outcome: no previous psychiatric history, no history or mental status examination findings of psychotic features, diagnosis of mood or adjustment disorder, no more than one psychotropic medication at baseline, good baseline functional status, fewer stressful events occurring during treatment, good baseline social support network, and good program attendance.

METHOD

Subjects

After appropriate human subject protection clearances were obtained, the medical records of 100 patients were retrospectively reviewed. Subjects consisted of all individuals (except for one whose chart could not be located) enrolled in the intensive treatment phase of the geriatric day treatment service after January 1, 1985, who were discharged before September 15, 1989.

To be enrolled in the geriatric day treatment service during the time covered by the study, patients had to be 60 years old or older (although those younger than 60 were considered on the basis of documented need), English-speaking, ambulatory, and able to provide their own transportation and lunch. They also had to have a

stable living situation, no substantial cognitive impairment, no overriding medical problems that would restrict their participation in the program, and no drug or alcohol problems requiring detoxification.

Geriatric Day Treatment Program

The program at our facility operates 3 days a week, but no individual patient attends more than twice a week. Patients are first admitted to an intensive treatment phase (2 days a week for approximately 3 months), followed by a modified treatment phase (2 days a week for approximately 3 months) and then an open-ended maintenance phase (1 day a week). Patients may be discharged at any point on the basis of clinical circumstances. Initially, patients undergo a psychiatric evaluation and examination and are assigned to a case manager (nurse, social worker, or occupational therapist). During the first few weeks of the program, patients receive an occupational therapy evaluation (including occupational history and assessment of current functional status), a home visit and assessment, a family assessment, and, if indicated, neuropsychological testing.

The mainstay of treatment is group therapy. Tasks and topics range from educational/didactic and concrete (time structuring) to more abstract and experiential (group psychotherapy). Patients receive individual attention from their case manager, but individual psychotherapy is not provided. However, individual or family psychotherapy as well as other ongoing treatments such as pharmacotherapy are recommended as indicated.

Instrumentation

For the retrospective chart review, a chart abstraction form and guidelines for its use were developed (these are available on request from D.A.P.). Members of a multidisciplinary geriatric partial hospitalization/day treatment research study group suggested which components of the process and outcomes of care should be assessed. The final version of the abstraction form consisted of 45 items and required about an hour to complete. Substantive areas of the form included demographic and clinical characteristics as well as social support network, functional status, and attendance record. Clinical progress was scored on an ordinal scale (most improved, improved, no change, worse) to be used as the outcome variable in statistical analyses.

A research assistant was trained to abstract the records according to written guidelines. The rater and the trainer independently abstracted the same five charts, discussed discrepancies, and addressed all questions with one of us (D.A.P.). Subsequently, another five charts were independently abstracted by the same two individuals; for these charts, interrater agreement was perfect.

Statistical Analysis

Data were analyzed by using SPSS-X (54). We used univariate and bivariate analyses to describe the study

TABLE 1. Demographic Characteristics of 100 Psychogeriatric Day Treatment Patients

Characteristic	Number of Patients
Gender	
Male	27
Female	73
Marital status	
Single/separated/divorced	22
Married	26
Widowed	52
Education	
Less than high school	21
High school or trade school	30
College	33
Insufficient data	16
Ethnic background	
White, not Hispanic	91
Other	9
Living site	
Private residence	77
Retirement hotel/home	20
Board and care	1
Insufficient data	2
Household composition	
Lives alone	37
Lives with spouse	26
Other	32
Insufficient data	5
Social network	
Family plus other support	56
Family support only	22
Other (nonfamily) support only	4
None/none noted	5
Referral source	
UCLA inpatient facility	37
Other	53

group and the level of program participation and bivariate and multiple regression analyses to examine predictors of program outcome. Because the outcome variable was ordinal and all hypotheses assumed an ordered effect, bivariate analyses were examined for an ordered effect. This was accomplished for five continuous independent variables (e.g., age, attendance) by one-way analyses of variance (ANOVAs) using linearity, and for 16 dichotomous independent variables (e.g., source of referral, social supports) by Kendall's tau statistic. Variables were also entered in multiple regression models (forward selection) in order to provide estimates of the unique contributions of individual independent variables to program outcome.

RESULTS

Characteristics of Patients

The mean age of the patients was 72.64 years (SD=7.01, range=56-87). The majority of patients were white, female, and living in a private residence; although almost 75% were widowed, single, separated, or divorced, only 37% lived alone (table 1). With regard to social network, records indicated that 93 patients had some sort of social support system available

TABLE 2. Medical and Psychiatric Characteristics of 100 Psychogeriatric Day Treatment Patients

Characteristic	Number of Patients
Positive psychiatric history	91
Suicide attempt within 1 year of admission	7
Psychotic symptoms within 1 year of admission	23
Past psychiatric hospitalization	61
Psychosocial stressors within 1 year of admission ^a	
None	59
One stressor	37
Two stressors	4
Medications	
Any medications	95
Multiple psychotropics	45
Antidepressants	65
Anxiolytics	47
Antipsychotics	18
Anticholinergics	4
Drugs for medical conditions	68
Mental status examination findings	
Cognitive impairment	13
Psychosis	4
Cognitive impairment and psychosis	1
Suicidal ideation	5
Diagnosis	
Depressive disorder only	47
Depressive disorder and other diagnoses	23
Bipolar disorder only	6
Bipolar disorder and adjustment disorder	1
Anxiety disorder only	3
Anxiety disorder and adjustment disorder	1
Anxiety disorder and personality disorder	1
Adjustment disorder only	9
Adjustment disorder and personality disorder	2
Other psychiatric disorder	7
Functional status	
Occupational therapy assessment (N=55)	
Good task performance skills	22
Good independent living skills	39
Good time management/socialization skills	10
Geriatric home assessment (N=68)	
Problem with self care	21
Problem with meals	10

^aDeath of a family member or close friend, major change in personal relationship, suicide attempt, psychotic symptoms (e.g., delusions, hallucinations), serious medical illness, psychiatric hospitalization, medical hospitalization, major change in financial status.

but did not consistently indicate whether support was actually provided. Sources of support other than family included friends, community involvement, and religious involvement and tended not to be noted unless they were indeed supportive.

The most commonly cited reasons for referral to the program were transition from hospitalization (42%), psychosocial needs such as time structuring, socialization skills, and occupational problems (38%), or as an alternative to hospitalization (12%). Our facility was the source of referral for 37 patients; 63 came from other referral sources.

At baseline, the majority of patients (70%) had a depressive disorder, either alone or in combination with another disorder (table 2). Psychotropic medications were being used by 86%, and more than half of these were receiving more than one. The 100 patients were

TABLE 3. Multiple Regression Models for Analysis of Clinical Status of 100 Psychogeriatric Patients at the End of Intensive Day Treatment^a

Variable Selected ^b	R ² and Adjusted R ²					R ² Change		
	R ²	Adjusted R ²	F	df	p	R ² Change	F	p
Model 1 (N=100)								
Number of group therapy sessions attended	0.26	0.26	35.23	1, 98	0.0000	0.26	35.23	0.0000
Stressful events during intensive treatment phase	0.33	0.32	23.89	2, 97	0.0000	0.07	9.49	0.0000
Number of social supports	0.38	0.37	19.95	3, 96	0.0000	0.05	8.41	0.002
Diagnosis of mood disorder	0.42	0.40	17.45	4, 95	0.0000	0.04	6.51	0.005
Model 2 (N=72)								
Functional status	0.10	0.09	7.97	1, 70	0.006	0.10	7.97	0.006
Diagnosis of mood disorder	0.21	0.18	8.95	2, 69	0.0004	0.10	9.02	0.004
Number of social supports	0.28	0.25	8.69	3, 68	0.0001	0.07	6.71	0.01

^aGroup therapy 2 days a week for approximately 3 months.

^bThe variables not selected were, in model 1, age, gender, widowhood, and history of psychosis, and in model 2, age, gender, widowhood, history of psychosis, stressful events during the intensive treatment phase, and number of group therapy sessions attended.

taking a mean of 2.71 medications each (SD=1.41). Not all patients underwent formal assessment of functional status (occupational therapy assessment and geriatric home assessment) because some patients left the program before those evaluations were made. To include all the patients for whom we had at least some information, we used the measures of task performance and self-care to create a composite variable (functional status) for analysis.

Course and Outcome

During the intensive treatment phase, the 100 patients attended a mean of 57.7 therapy groups (SD=34.2, range=2-124). At the end of this phase, some degree of improvement was observed in 57% of the patients: 31 had improved moderately or markedly and 26 had improved minimally. Thirty patients showed no change, and 13 were worse. Forty-two percent experienced one or more psychosocial stressors (table 2) during intensive treatment: 26 experienced one, 12 experienced two, and four experienced three stressors. No psychosocial stressors were noted for 58 of the patients. More than half of the patients (N=58) went on to the modified treatment phase; 37 patients were discharged early and five were discharged at the end of this treatment phase. The patients who did not go on to the modified treatment phase had significantly poorer clinical outcomes at the end of the intensive treatment phase (Kendall's tau=-0.54, df=3, p<0.0001).

In general, the quality of the data for the modified treatment phase was poor, and only general trends could be reliably identified. Almost one-fourth of the patients who went on to the modified treatment phase improved in clinical status after completion of the intensive treatment phase, and about 40% were unchanged or worse. For the remaining patients, insufficient data were available to judge progress. About two-thirds experienced one or more psychosocial stressors during the modified treatment phase. The mean number of groups attended during the modified treatment phase was 59.3 (SD=60.7) (range=0-206).

About one-fourth of the patients were discharged

from the geriatric day treatment service because of hospitalization (13 for psychiatric hospitalization and 13 because of medical hospitalization).

Predictors of Outcome

The bivariate analyses identified eight variables associated with outcome at the p<0.05 level: diagnosis of mood disorder (Kendall's tau=-0.25, df=3, p=0.004), history of psychosis (Kendall's tau=0.16, df=3, p=0.04), functional status (Kendall's tau=0.28, df=3, p<0.01), presence of social support other than family (Kendall's tau=-0.38, df=3, p<0.0001), number of areas of social support (ANOVA linearity F=23.37, df=1, 2, p<0.0001), the occurrence of stressful events during the intensive treatment phase (Kendall's tau=0.19, df=3, p=0.02), the number of stressful events during the intensive treatment phase (ANOVA linearity F=4.12, df=1, 2, p<0.05), and attendance (ANOVA linearity F=42.97, df=1, 2, p<0.0001). Each was associated in the hypothesized direction.

Three of the hypothesized predictors were not associated with outcome: number of medications (ANOVA linearity F=0.01, df=1, 2, p=0.93), psychotic symptoms on mental status examination (Kendall's tau=0.05, df=3, p=0.31), and psychiatric history (only six out of 97 patients did not have a psychiatric history, so further analysis was precluded).

Table 3 summarizes two multiple regression models with clinical status at the end of the intensive treatment phase as the dependent variable. The first model achieved statistical significance and explained about 40% of the variance in outcome. It contained only variables for which data on all 100 patients were available (i.e., it did not include the functional status measure) and selected all but one (history of psychosis) of the variables that had been identified in the unadjusted bivariate analyses. The second model included only the 72 patients for whom information on functional status was available. All but one of the 28 patients lacking such data were among those who were discharged early. The second model also achieved significance and explained about 25% of the observed variance in clinical

cal outcome for these 72 patients. Examination of each step of the analyses confirmed that, in general, independent variables identified in the initial bivariate analysis made additive and independent contributions. The only exception was in the second model, in which functional status and number of social supports shared variance.

DISCUSSION

In terms of clinical population, our results are similar to those of Eskew et al. (43) and Wagner (44). For outcomes achieved, our results are remarkably similar to those of Eskew et al. and to results of studies in younger patient populations. The findings suggest that a majority of patients improve with day treatment and that slightly more than one out of 10 gets worse. Further, it appears 1) that treatment programs such as these should monitor patient characteristics, processes and outcomes and that it is feasible to do so, 2) that partial hospitalization programs for the elderly (with some exceptions, such as Department of Veterans Affairs programs) may tend to treat widowed, depressed women, and 3) that some of the factors associated with outcome are potentially modifiable (functional status, social network, and attendance). The next generation of studies should look specifically at the effects on outcomes of modifying these factors through interventions.

Consistent with the nonexperimental and retrospective nature of the study, a number of considerations apply with regard to the baseline characteristics associated with outcome. The relative homogeneity of our sample would tend to obscure associations of baseline characteristics with outcome; this may explain why some characteristics (e.g., psychiatric history, mental status examination findings, psychotropic medication status) were not associated with outcome, while underscoring the importance of those variables which were associated with outcome.

For social support, the available data did not allow us to discriminate between family being available and family actually being supportive. Thus, our results suggest that social support other than family is the crucial element associated with outcome, which may be an artifact of the study.

For functional status, our program admission criteria select for generally high-functioning patients, which would tend to narrow the field and obscure statistical associations. Furthermore, information was not available on this variable for about one-fourth of the patients in our study, notably those who were discharged early (and who also had unfavorable outcomes). Because such patients are likely to have had functional status impairments that interfered with attendance in our program, their absence from the analysis would tend to weaken the observed association between functional status and outcome. Thus, the observed effect is compelling preliminary evidence for the importance of baseline functional status in a day treatment program.

With regard to stressful events during treatment, our results suggest that such events are associated with a less favorable outcome, in keeping with clinical lore as well as research findings in this area (55-57). However, our results may be a consequence of having relatively short-term outcomes that were sensitive to untoward recent events rather than indicative of unfavorable response to treatment.

Our data indicate that the number of therapy groups attended was positively associated with clinical outcome for the intensive treatment phase. Although this is consistent with the individual psychotherapy literature (58), it may reflect a selection bias rather than a treatment effect in our patients because sicker patients would be more likely to have poor attendance and unfavorable outcome.

With regard to outcomes achieved, our estimates were derived from clinician notes and may be biased in the direction of emphasizing improvement; in any event, it may be that the improvement was due to factors other than the day treatment services provided (e.g., other ongoing treatments such as psychotropic medication, for which data other than baseline were lacking). Nevertheless, the data illustrate some general patterns that may be worthy of further study under more optimal conditions. Given the current climate of cost containment and the increasing scrutinization of conventional inpatient treatment (59), with its implication for the development of rational alternatives, further investigations of partial hospitalization for elderly psychiatric patients are recommended.

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Familial Aggregation of Female Sexual Orientation

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Objective: The purpose of this study was to determine whether female homosexuality is familial and whether it is cofamilial with male homosexuality. **Method:** Subjects included 84 homosexual and 79 heterosexual female probands recruited through newspaper advertisements. Probands were asked about their siblings' sexual orientations and were asked for permission to contact siblings to confirm their reports. **Results:** The authors were able to contact 60% of eligible siblings, and the information they provided about their sexual orientations confirmed that probands' reports were highly accurate. Homosexual probands had a significantly higher proportion of homosexual sisters according to four criteria for rating siblings' sexual orientations. Homosexual probands also had a higher proportion of homosexual brothers; however, this difference was not significant. **Conclusions:** Female homosexuality appears to be familial. Further research is required to resolve the question of whether female and male homosexuality are cofamilial.

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Although research during the past decade has begun to illuminate the etiology of male sexual orientation (1-5), the causes of female sexual orientation remain largely unknown. At the nascent stages of research, family studies may provide particularly useful information. For instance, a finding that a trait is familial supports the desirability of studying genetic and familial environmental factors by using methodologies from, for example, behavioral genetics and family dynamics. A second issue that can be explored through family studies concerns the etiological overlap between different phenotypes. If two phenotypes are etiologically related and are both familial, then they may be expected to co-occur in the same families. Determining whether female and male homosexuality are cofamilial would inform theories of both phenomena.

There have been at least three family studies relevant to female homosexuality. Bell et al. (6) found that female homosexuals reported more homosexual siblings than did female heterosexuals; unfortunately, results

were not broken down by sex of siblings. Bailey et al. (7) found that female homosexual probands suspected more of their sisters to be homosexual than did heterosexual probands. However, the number of sisters known to be homosexual did not differ. The most systematic family study to date was reported by Pillard (8), who was able to contact a large proportion of the siblings to verify probands' reports. He found 25% of 60 sisters of homosexual female probands to be homosexual (or bisexual), compared to 11% of 53 sisters of heterosexual female probands. Regarding the relationship between male and female homosexuality, both Bailey et al. and Pillard found some evidence that male and female homosexuality are somewhat cofamilial, since female homosexual probands reported more homosexual brothers than female heterosexual probands. Thus, the existing literature suggests that female homosexuality may be familial and that it may be related to male homosexuality. The purposes of the study reported here are to provide the most powerful test of familiarity of female homosexuality to date and to investigate the cofamiliarity of male and female homosexuality.

METHOD

Subject Recruitment and Interviews

Female homosexual (including bisexual) and heterosexual probands were recruited from several sources. Homosexual probands were recruited primarily through advertisements in lesbian and gay publications in Chicago. The ads specified that desired subjects were "lesbian or bisexual women" between the ages of 25 and

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40 and that the study concerned "personality, attitudes, interests, and sexual behavior." The age limits were used to maintain a degree of homogeneity in the study group and to ensure that the probands' sexual orientations were relatively stable. No mention was made of our interest in familiarity of female homosexuality. Advertisements were also displayed in several lesbian bars, and a sign-up sheet was posted at a conference for lesbians. Female heterosexuals were recruited through an advertisement in an alternative newspaper that was identical to the advertisement for homosexuals except that "heterosexual women" replaced "lesbian or bisexual women." In order to be selected, a proband must have had at least one brother aged 18 years or older or one sister aged 25 or older. The minimum ages were chosen so that a sibling who had reached the relevant age would be reasonably likely to have established a stable sexual orientation. The sex differences reflect the fact that, on average, men accomplish this earlier than women (9–11).

Subjects who met the inclusion criteria were scheduled for a 1–2-hour interview at a university family studies laboratory. All interviews were conducted with informed consent. The session included questions concerning the proband's sexual orientation and siblings' sexual orientations. After completion of the interview, each proband was asked for permission to contact her siblings who exceeded the minimum ages. Probands inspected the questionnaire to be sent and were assured that the true nature of the study would not be divulged to siblings.

The premise of the cover letter to siblings was that they were being asked to participate in a general behavioral genetics study of personality, attitudes, and behavior. Five questions regarding sexual orientation were embedded within over 100 other multiple-choice items about social attitudes, personality, and family relationships. Questionnaires were sent to consenting probands' eligible siblings. If siblings did not respond within approximately 1 month, attempts were made to contact them by telephone. Efforts to gain their cooperation were halted only if at least two mailings of the questionnaire were unsuccessful and 1) no telephone number was available for the sibling, 2) repeated telephone calls were unsuccessful, or 3) the sibling declined to participate.

This procedure resulted in 163 proband interviews. Probands were assigned to either the homosexual or the heterosexual group according to self-identification: those who considered themselves either lesbian/homosexual or bisexual were assigned to the homosexual group; those who considered themselves heterosexual were assigned to the heterosexual group. The homosexual group consisted of 84 probands, and the heterosexual group consisted of 79 probands. Of the homosexual probands, 61 described themselves as lesbian/homosexual, and 23 described themselves as bisexual. Separate Kinsey ratings (9, 10) were obtained for adult fantasy and behavior. Kinsey scores of 0 and 1 are generally considered to represent heterosexuality, 2–4 to repre-

TABLE 1. Characteristics of 84 Homosexual and 79 Heterosexual Women

Item	Homosexual Women	Heterosexual Women
Age (years)		
Mean	32.4	32.4
SD	4.9	4.9
Education (years) ^a		
Mean	5.9	6.0
SD	0.7	0.8
Ever married ^b		
N	14	24
%	17	30
Kinsey adult fantasy score		
Mean	4.3	0.5
SD	1.4	0.7
Kinsey adult behavior score		
Mean	4.2	0.2
SD	1.6	0.5
Number of sisters	105	84
Number of brothers	118	88

^a3=Some high school, 4=high school graduate, 5=some college, 6=college graduate, 7=postgraduate degree.

^b $\chi^2=4.3$, $df=1$, $p<0.05$.

sent bisexuality, and 5 and 6 to represent predominant homosexuality. Both the mean Kinsey fantasy and behavior scores of the homosexual probands (mean=4.3, SD=1.4, and mean=4.2, SD=1.6, respectively) were in the upper part of the bisexual range. In contrast, the heterosexual probands' respective scores (mean=0.5, SD=0.7, and mean=0.2, SD=0.5) were well within the heterosexual range.

Subject characteristics are given in table 1. Homosexual and heterosexual probands did not differ significantly in mean age or educational level. A significantly smaller proportion of homosexual women had ever been married; however, this percentage was relatively small in each group (17% of homosexuals versus 30% of heterosexuals; $p<0.05$). This reflects the fact that the heterosexual probands were somewhat unrepresentative of women from the general population, probably because of the alternative nature of the publication that included the advertisements.

Homosexual probands had a total of 223 siblings of interest, and heterosexual probands had a total of 172 (table 1). Permission was granted to contact 237 (60%) of eligible siblings. The percentage of siblings for whom homosexual probands gave consent to contact (58%) was less than the analogous percentage for heterosexual probands (67%) ($\chi^2=3.5$, $df=1$, $p=0.06$). Siblings returning questionnaires totaled 215 (122 sisters and 93 brothers), representing 54% of the entire group of siblings and 91% of those for whom probands gave consent to contact. The percentages of siblings who returned questionnaires did not differ according to the sexual orientation of the proband.

Assessment of Siblings' Sexual Orientation

The sexual orientation of siblings was assessed in two ways. First, probands were asked whether they believed

TABLE 2. Sexual Orientation Ratings of Siblings of 84 Homosexual and 79 Heterosexual Female Probands by Siblings and Probands

Rating of Sibling by Proband	Self-Rating by Sibling							
	Heterosexual		Bisexual		Homosexual		Could Not Contact	
	M	F	M	F	M	F	M	F
Proband at least virtually certain of sibling orientation								
Heterosexual	82	104	1	3	0	0	90	57
Bisexual	1	0	0	2	0	1	2	1
Homosexual	0	0	0	0	2	6	4	1
Proband less confident of sibling orientation	7	6	0	0	0	0	17	8

their siblings' sexual orientation to be heterosexual, homosexual, or bisexual. In addition, probands were asked how certain they were about their assessment, using the following scale: "Completely certain" indicated that the sibling had told the proband his or her orientation; "virtually certain" meant that the proband felt quite sure, but that this was based on behavior alone; "suspect, but not sure" meant that the proband had some reason for making a guess but felt appreciable uncertainty; "very uncertain" meant that the proband could do little more than guess. In addition, those siblings who could be contacted were asked directly to rate themselves as heterosexual, bisexual, or homosexual/lesbian/gay. Siblings also gave ratings of their attraction to men and women, respectively, as well as separate Kinsey ratings for adult fantasy and behavior.

Siblings' self-ratings of sexual orientation were used when available. However, for a large percentage of siblings these data were lacking. We had reason to believe, on the basis of past studies of male homosexuality (2, 4) and female homosexuality (personal communication from R.C. Pillard, M.D.), that probands would generally be quite accurate in assessing their siblings' sexual orientation provided that the proband expressed a high level of confidence. This was, in fact, confirmed for those siblings for whom both ratings were available, as is evident in table 2. In those 202 cases in which a proband was at least virtually certain about her sibling's orientation, the prediction of heterosexual versus not heterosexual (i.e., either homosexual or bisexual) was incorrect only five times, for an overall accuracy rate of 97.5%. The kappa reliabilities associated with table 2 (excluding the "proband less confident of sibling orientation" row and the "could not contact" column) were 0.80 for sisters and 0.66 for brothers. In one case in which a heterosexual proband claimed that her brother was bisexual, the brother said that he was heterosexual. In four cases in which probands believed that their siblings were heterosexual, the siblings said that they were bisexual. Three of these four (two sisters and a brother) were siblings of homosexual probands.

Given the high degree of accuracy for confirmable cases when probands expressed a high degree of certainty, it was decided that without a sibling's self-rating, the proband's assessment of her sibling's sexual orientation would be used provided that the proband was at least virtually certain. If a sibling's self-rating

was unavailable and the proband was less confident, that case was omitted from analyses of sexual orientation. Sexual orientation ratings were available for 370 of the 395 eligible siblings.

RESULTS

The rates of homosexuality (including bisexuality) among sisters of homosexual and heterosexual probands are presented in table 3 according to four separate criteria. Because our hypothesis for sisters is strongly unidirectional (i.e., sisters of homosexual probands should have a higher rate than sisters of heterosexual probands), the probability levels reported later in the paper are one-tailed. The first criterion, as explained earlier, included all siblings who indicated that they were either homosexual or bisexual, as well as all siblings who did not participate in the study but whose sexual orientations were rated by probands, with at least certainty, as either heterosexual or bisexual. According to this criterion, 12.1% of the sisters of homosexual probands were rated homosexual, compared to 2.4% of the sisters of heterosexual probands ($p < 0.05$). One homosexual proband had three homosexual sisters, two had two, and five had one each; the two homosexual sisters of heterosexual probands were from separate families.

The other criteria for rating siblings as homosexual could be met only by siblings who participated in the study. The second criterion was simply rating oneself as either homosexual or bisexual. By this criterion, 15.4% of sisters of homosexual probands indicated that they were homosexual, compared to 3.5% of sisters of heterosexual probands ($p < 0.05$).

The third criterion for homosexuality used Kinsey scores (9, 10) for adult sexual fantasy, which were available only for siblings who participated in the study. This criterion for homosexuality included siblings with Kinsey adult fantasy scores of 2 or greater. A Kinsey score of 2 on this scale indicates that during adulthood most of one's sexual fantasies have concerned the opposite sex but that one has had more than an occasional fantasy about the same sex. This more lenient criterion was included in order to compare our results with those of Pillard (8). By this criterion, 21.2% of the sisters of homosexual probands were rated

TABLE 3. Rates of Homosexuality in Siblings of 84 Homosexual and 79 Heterosexual Female Probands

Sibling Group	Criterion for Homosexuality ^a											
	1: Proband or Sibling Report			2: Sibling Report			3: Kinsey Adult Fantasy Score			4: Homosexual Feelings		
	Total	Siblings Rated		Total	Siblings Rated		Total	Siblings Rated		Total	Siblings Rated	
		Homosexual	%		Homosexual	%		Homosexual	%		Homosexual	%
N	N	%	N	N	%	N	N	%	N	N	%	
Sisters of homosexual probands	99	12	12.1 ^b	65	10	15.4 ^c	66	14	21.2 ^d	66	23	34.8 ^e
Sisters of heterosexual probands	83	2	2.4	57	2	3.5	58	3	5.2	58	8	13.8
Brothers of homosexual probands	110	8	7.2 ^f	49	3	6.1	49	3	6.1	49	4	8.2
Brothers of heterosexual probands	81	1	1.2	44	0	0.0	44	1	2.3	43	1	2.3

^aThe four criteria for rating a sibling homosexual are as follows. Criterion 1: either the sibling participated and indicated a homosexual (or bisexual) orientation or, if the sibling did not respond, the proband was at least virtually certain that the sibling was homosexual; criterion 2: sibling participated and indicated a homosexual orientation; criterion 3: sibling participated and endorsed a Kinsey adult fantasy score of 2 or more; criterion 4: sibling participated and admitted to finding the idea of homosexual activity sexually arousing. Percents for criteria 2-4 were computed by using the number of relevant subjects for whom data were available in the denominator.

^b $\chi^2=6.0$, $df=1$, $p<0.05$.

^c $\chi^2=4.8$, $df=1$, $p<0.05$.

^d $\chi^2=6.7$, $df=1$, $p<0.05$.

^e $\chi^2=7.3$, $df=1$, $p<0.01$.

^f $p=0.08$, Fisher's exact test.

homosexual, compared to 5.2% of the sisters of heterosexual probands ($p<0.05$).

The final and least stringent criterion for homosexuality included all siblings who admitted that they found the idea of homosexual activity at least somewhat sexually exciting. According to this criterion, 34.8% of the sisters of homosexual probands were homosexual, compared to 13.8% of the sisters of heterosexual probands ($p<0.01$). (Group sizes differ slightly among the different criteria, since some siblings did not respond to all questions.)

The rates of homosexuality in brothers are also presented in table 3. Because brothers of female homosexual probands might conceivably be predicted to be either more or less likely to be homosexual, the probability levels were two-tailed. By the first criterion, homosexual probands had a marginally significant proportion of homosexual brothers compared to heterosexual probands ($N=8$ of 110 versus $N=1$ of 81; $p=0.08$, Fisher's exact test). Unfortunately, relatively few brothers who met any of the criteria for homosexuality participated in the study (the maximum being subjects who met the fourth criterion). Therefore, although more brothers of homosexual probands were rated as homosexual by the final three criteria than were brothers of heterosexual probands (table 3), none of these differences approached significance.

One possible concern regarding the analyses reported earlier is that they include multiple siblings of the same proband. This may violate the assumption of independent observations required for the chi-square test. If so, the significance tests reported earlier are somewhat liberal. To control for this possibility, analyses were also performed in which each proband accounted for only one observation. Data were analyzed by a series of eight contingency tables, one for each method of assessing siblings' sexual orientations, separately for brothers

and sisters. The data comprising these contingency tables, and their associated probabilities, are reported in table 4. The frequencies in the tables for sisters represent, respectively, the number of homosexual probands with at least one homosexual sister, the number of homosexual probands with at least one heterosexual but no homosexual sister, and the two similar frequencies for heterosexual probands. Frequencies for brothers were computed analogously. Consistent with the results reported earlier, by all four of the criteria for assessing homosexuality in sisters, homosexual probands were significantly more likely than heterosexual probands to have a homosexual sister. Only one of the four analyses for brothers was significant. Homosexual probands were more likely to have a homosexual brother as assessed by either sibling or proband report.

DISCUSSION

Results of the present study suggest that female homosexuality is familial. We found significant familiarity under four definitions of homosexuality despite the widely varying rates that emerged with the different criteria, ranging from 12.1% to 34.8% for sisters of homosexual probands and from 2.4% to 13.8% for sisters of heterosexual probands. However, there are at least four potential methodological problems that could have led to spurious findings of familiarity. First, because of the way they were recruited, the homosexual probands were self-selected for openness and thus may have been particularly knowledgeable about their siblings' sexual orientations. However, heterosexual probands were also self-selected for openness, since they also volunteered for a study of sexuality. Future studies that avoid this possible methodological confound, for example, by random sampling, are clearly desirable.

TABLE 4. Number of 84 Homosexual and 79 Heterosexual Female Probands With Homosexual Versus Heterosexual Siblings

Sibling Sex	Criterion for Homosexuality	Number of Homosexual Probands		Number of Heterosexual Probands		p ^a
		With at Least One Homosexual Sibling of Designated Sex	With at Least One Heterosexual but No Homosexual Sibling of Designated Sex	With at Least One Homosexual Sibling of Designated Sex	With at Least One Heterosexual but No Homosexual Sibling of Designated Sex	
Female	1: proband or sibling report	8	50	2	50	0.05
	2: sibling report	8	33	2	38	0.04
	3: Kinsey adult fantasy score of 2 or more	12	30	3	38	0.01
	4: admission of homosexual feelings	19	23	8	33	0.02
Male	1: proband or sibling report	8	58	1	56	0.04
	2: sibling report	3	31	0	35	0.11
	3: Kinsey adult fantasy score of 2 or more	3	31	1	34	0.36
	4: admission of homosexual feelings	4	30	1	35	0.19

^aFisher's exact test. Probabilities for females are one-tailed; probabilities for males are two-tailed.

Second, if homosexuals with homosexual sisters were more likely than heterosexuals with homosexual sisters to cooperate, this could have led to a spurious difference. However, in our advertisements for subjects, we did not mention either familiarity or siblings. Thus, it is difficult to imagine an alternative mechanism that would cause this kind of ascertainment bias, although it cannot be definitively ruled out.

A third issue concerns the sisters who did not participate in the study. A substantial percentage (35%) of sisters did not provide information, and it is mathematically possible that if they had, female homosexuality would not have appeared to be familial. However, this seems unlikely. Probands' reports that could be verified were quite accurate. The three errors that probands made in assessing their sisters' orientations occurred when probands designated them as heterosexual but the sisters considered themselves bisexual. Two of these three were sisters of homosexual probands, which suggests that the different rates of homosexual sisters resulted neither from homosexuals' overestimation nor from heterosexuals' underestimation of familial homosexuality.

Finally, it is conceivable that the heterosexual probands may not have been representative of the general heterosexual population, having a lower number of homosexual siblings. These women were certainly not representative of the general population in at least two respects: a high average level of education and a low marriage rate. As previously noted, this is due to the nature of the publication from which they were recruited, which is oriented toward educated, politically liberal, nontraditional professionals with high incomes. It seems unlikely that these characteristics should diminish familial homosexuality. Furthermore, the rate of homosexuality found among the sisters of the heterosexual probands, according to the strictest criteria, 2.4%, is close to Gebhard's estimate of 1.5% for predominant female homosexuality in the general population (12).

Our results concerning the familiarity of female homosexuality are similar to those obtained by Pillard (8), who used a similar methodology. He used a criterion of Kinsey scores (combined fantasy and behavior) of 2 or greater. The rate that he reported in the sisters of homosexual probands (25%) was similar to that which we obtained using our comparable criterion (Kinsey fantasy scores of 2 or greater; 21%). Although Pillard found a somewhat higher rate in the sisters of heterosexuals (11% versus 5% in the present study), the difference was not significant.

Because of the high accuracy of probands' reports and the substantial percentage of sisters (35%) for whom only these were available, the most accurate figures are probably those which also used proband reports when sibling self-reports were not available: 12.1% for sisters of homosexual probands and 2.4% for sisters of heterosexual probands. However, we emphasize that our rates varied widely according to the definition used. Our preferred figures apply to an identification as either homosexual or bisexual, which was the most conservative criterion examined. Assuming these figures are accurate, what implications do they have? Because the 12.1% rate implies that the large majority of sisters of female homosexuals are heterosexual, one might be tempted to conclude that familial factors are of minor etiological importance. However, a multifactorial model of transmission, in which one's sexual orientation depends on a continuous underlying etiological dimension that is influenced by many genetic or environmental factors each of small effect, can reconcile powerful familial factors with rates such as ours (13, 14). Further research is needed to determine the magnitude of familial etiological factors.

Results regarding the rate of homosexuality in brothers were less clear than for sisters. Under one criterion there was a trend for a higher rate of homosexuality among brothers of homosexual probands (7.2% versus 1.2%). Assuming that this difference is a real

one, i.e., that male and female homosexuality are cofamilial, do the results tell us anything about the extent of their etiological overlap? It is noteworthy that the rate of homosexuality in brothers of homosexual probands was approximately half that in sisters, although this difference was not significant. Pillard (8) obtained a similar ratio (9% of brothers versus 25% of sisters). This ratio is markedly less than one would expect if male and female homosexuality were determined by identical causes because Gebhard estimated the base rate of male homosexuality as more than twice that of female homosexuality (12). Thus, the available evidence suggests that even if male and female homosexuality are somewhat cofamilial, they are also somewhat independent.

CONCLUSIONS

Female homosexuality appears to run in families. The factors responsible for the familiarity, however, remain unknown. The findings of this study support the desirability of using more sophisticated methodologies, such as twin and adoption studies, to unravel the effects of genes and shared environment on female sexual orientation. The familial relationship between female and male homosexuality remains unclear. Further studies, using larger groups of subjects, will be needed to resolve this question.

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Are Schizophrenia and Affective Disorder Related? Preliminary Data From a Family Study

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***Objective:** Most investigators presume that schizophrenia and affective disorder are separate diseases. Others have proposed alternatives to this Kraepelinian view. These alternatives were addressed by preliminary analyses of data from a family study of psychopathology. **Method:** The authors identified 1,895 first-degree relatives of 166 patients with DSM-III schizophrenia, 71 patients with affective disorder, and 85 medical comparison probands; 949 relatives were blindly diagnosed. **Results:** The risks for schizophrenia and affective disorder (unipolar melancholia and bipolar disorder combined) were significantly higher in the relatives of the schizophrenic probands and the relatives of the probands with affective disorder than in the relatives of the comparison probands. The morbid risk for nonmelancholic depressions was not significantly higher. Among the relatives of the schizophrenic probands, the risk for affective disorder was highest among the relatives of the patients with "core" schizophrenia, who were younger at illness onset, had chronic illness, had severe emotional blunting, and showed few affective features. **Conclusions:** Despite limitations, these preliminary analyses, consistent with other studies, suggest some familial relationship between schizophrenia and severe forms of affective disorder.*

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Most investigators presume that schizophrenia and affective disorder are biologically distinct. Evidence supporting this Kraepelinian perspective primarily derives from family and twin data (1, 2). Others have proposed that the two psychoses are poles of a continuum on which schizophrenia is the severe form and schizoaffective disorder is intermediate (3). Some suggest that schizophrenia and bipolar affective disorder, although distinct, are each related to unipolar depression and schizoaffective disorder, which are related to each other (4). These alternatives also derive from

family and twin data, but the studies supporting the Kraepelinian perspective exceed those which do not.

Despite shortcomings (1, 5), studies generally have shown that schizophrenia and affective disorder are familial. Nevertheless, there is evidence that these psychoses co-occur in some families. Tables 1 and 2 display some of that evidence. The studies in table 1 (4-9) showed a high morbid risk for affective disorder in the first-degree relatives of schizophrenic subjects. The studies in table 2 (4, 6, 9-14) showed a high morbid risk for schizophrenia in the first-degree relatives of affectively ill patients. These reports are supported by earlier work (15-20) and, coupled with those questioning the unipolar/bipolar dichotomy (6, 21, 22), suggest that the present classification of psychosis may need modification.

Some twin data also fail to fit the present nosology (23, 24). For example, the study by Slater and Shields (23) provides support for a genetic predisposition to schizophrenia with concordance rates for monozygotic and dizygotic twins of 65% and 14%, respectively. Nevertheless, the prevalences of affective disorder and schizophrenia were similar among the siblings of the schizophrenic index twins in that study, and among their parents affective disorder was more prevalent than schizophrenia. Co-occurrence is best illustrated by the reanalysis of the Maudsley twin data by Farmer et al. (24), who used the DSM-III criteria. Of 24 pairs of monozygotic twins in which at least one member was

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TABLE 1. Studies of Affective Disorder in Relatives of Schizophrenic Probands

Study	Proband Diagnosis	Morbidity Risk in First-Degree Relatives ^a	
		Diagnosis	Risk (%)
Tsuang et al. (6), (1980)	Schizophrenia	Bipolar disorder	2.4
	Mania	Bipolar disorder	5.3
	Depression	Bipolar disorder	3.0
	Normal	Bipolar disorder	0.3
Mendlewicz et al. (7), 1980	Schizophrenia	Bipolar disorder	8.6
Abrams and Taylor (5), 1983	Schizophrenia	Total bipolar and unipolar disorder	6.9
Guze et al. (8), 1983	Schizophrenia	Unipolar disorder	8.1
Gershon et al. (4, 9), 1988, 1982	Schizophrenia	Bipolar disorder	1.3
	Normal	Unipolar disorder	14.7
		Bipolar disorder	0.8
		Unipolar disorder	6.7
	Chronic psychoses (drugs)	Unipolar disorder	18.7

^aBase rate approximately 3% to 6%, depending on depressions that are included (see text).

originally diagnosed as schizophrenic and in whom the original concordance rate for schizophrenia was 42%, seven pairs each had one member with a schizophrenic syndrome and the other with an affective syndrome. Farmer et al. concluded that the problem lay with the DSM-III criteria. However, it is equally plausible that their data indicate some shared liability.

Despite these findings, the family study data are not interpreted as indicating substantial familial co-occurrence (1, 2). However, in some "negative" studies (13, 25), co-occurrence was observed but not addressed. In others (26, 27), no affective disorder was reported even though base rates should have been observed, suggesting that the investigators may not have looked for co-occurrence. Other negative studies (28, 29) had methodological problems that weaken the data. Nevertheless, some studies (30, 31) had good methods and adequate numbers of subjects and no significant co-occurrence was observed.

Problems with the Kraepelinian dichotomy also include 1) the unexpected high prevalence (25%) of mixed, schizoaffective psychoses; 2) failure to clearly distinguish psychotic subjects by their clinical features; 3) outcomes that fall along a continuum; 4) responsiveness to similar treatments (particularly for neuroleptics and ECT, but also for lithium) (32); 5) the fact that some patients experience early episodes of affective disorder followed by episodes of schizophrenia (about 10%), and vice versa (about 5%); and 6) the inability of laboratory measures, while delineating psychotic from non-psychotic patients, to clearly differentiate psychotic groups. Associated abnormalities in brain structure and function are not pathognomonic, and many are observed with similar frequencies in the two psychoses. The degree of overlap among studies of biological variables led Meltzer (33) to conclude that a continuum

TABLE 2. Studies of Schizophrenia in Relatives of Affectively Ill Probands

Study	Proband Diagnosis	Morbidity Risk in First-Degree Relatives ^a	
		Diagnosis	Risk (%)
Smeraldi et al. (10), 1977	Unipolar disorder	Schizophrenia	2.2
Angst et al. (11), 1980	Bipolar disorder	Schizophrenia	2.5
		Siblings	4.8
		Offspring	1.9
		Total	1.5
Scharfetter and Nusperli (12), 1980	Bipolar disorder	Schizophrenia	3.3
	Schizophrenia	Schizophrenia	8.9
Tsuang et al. (6), 1980	Mania	Schizophrenia	3.2
	Depression	Schizophrenia	1.7
	Schizophrenia	Schizophrenia	5.5
	Normal	Schizophrenia	0.6
Kendler et al. (13), 1985	Manic-depressive illness (psychotic)	Schizophrenia	4.3
		Manic-depressive illness	20.0
Kendler et al. (13, 14), 1985, 1986	Bipolar disorder (psychotic)	Schizophrenia	2.5
		Schizoaffective disorder	1.4
	Normal	Atypical psychosis	2.5
		Schizophrenia	1.0
		Schizoaffective disorder	1.0
		Atypical psychosis	0.1
Gershon et al. (4, 9), 1988, 1982	Bipolar disorder	Schizophrenia	0.3
	Unipolar disorder	Schizophrenia	0.0
	Schizoaffective disorder (acute)	Schizophrenia	4.9

^aBase rate between 0.4% and 0.6%.

was a reasonable interpretation of the data. One of us (34) has reviewed these issues in detail.

Therefore, empirical support for the present nosology is not without challenge, and further study of the discreteness of the psychoses seems warranted. We had the opportunity to do this and present here our preliminary findings on the familial relationship between schizophrenia and affective disorder.

METHOD

Psychiatric Probands

We selected psychiatric probands from acute and chronic psychiatric wards and clinics of a Veterans Affairs hospital. The probands were male, were age 18 or older, spoke English, and signed informed consent statements. Prospective probands were screened to eliminate subjects with recent alcoholism or drug abuse or clinical evidence of coarse brain disease (e.g., seizures, head trauma). The screening criteria for alcoholism were 1) alcohol abuse or dependence not primary diagnosis, 2) index episode not due to alcohol abuse or dependence, 3) no alcohol abuse or dependence during the year before entry into the study, and 4) no physical

or behavioral evidence of chronic alcohol abuse. The screening criteria for drug abuse were 1) drug abuse or dependence not primary diagnosis, 2) no drug dependence of any kind, 3) no use of hallucinogenic drugs (e.g., LSD, mescaline, PCP; use of cannabis was permitted), 4) index episode not due to drug abuse, and 5) no drug abuse during the prior year. The patients were then interviewed by two research psychiatrists using a semistructured instrument based on the Schedule for Affective Disorders and Schizophrenia—Lifetime Version (SADS-L) (35), the Schedule for the Assessment of Negative Symptoms (36) and Schedule for the Assessment of Positive Symptoms (37), an emotional blunting scale (38), and items for symptom intensity and duration assessing specific DSM-III criteria. The interview information was confirmed from the patients' charts and by the treating physicians and nurses. A DSM-III diagnosis of schizophrenia or affective disorder was required for entry into the study. We further divided the schizophrenic subjects into the following diagnostic subgroups.

1. *Core schizophrenia.* Emotional blunting (loss of emotional expression and avolition); no past or present affective episode or prominent affective features; clear consciousness; syndrome not secondary.

2. *Noncore schizophrenia.* No loss of emotional expression or personality deterioration, but patient could be avolitional; no past or present affective episode or prominent affective features; clear consciousness; syndrome not secondary.

3. *Schizoaffective disorder.* No loss of emotional expression or personality deterioration, but patient could be avolitional; definite past or present episode of mania or depression meeting DSM-III criteria but shorter in duration than nonaffective positive features of psychosis; clear consciousness; syndrome secondary.

The patients with core schizophrenia were chronic, deteriorated patients with no past or present affective disorder. Many had been hospitalized for decades (mean illness duration=29.47 years, SD=11.43). The patients with noncore schizophrenia had chronic psychoses with positive symptoms but no affective episodes; many had prolonged hospitalizations (mean illness duration=23.43 years, SD=9.57). Although they did not have personality deterioration, they never returned to premorbid functioning. Separation of emotional blunting into loss of expression and avolition was based on factor analytic data (38) indicating two orthogonal dimensions. The schizoaffective patients had chronic or subchronic psychotic affective disorder and, despite substantial amelioration of many positive features and sparing of personality, remained avolitional with moderate or severe loss of social or employment functioning (mean illness duration=24.47 years, SD=10.64). The three schizophrenic subgroups did not differ significantly from each other in illness duration.

The DSM-III and DSM-III-R criteria for schizophrenia differ somewhat. However, although DSM-III-R reduces the number of patients diagnosed as schizophrenic by 10%, patients with DSM-III schizophrenia

and patients with DSM-III-R schizophrenia differ only in the signs and symptoms reflecting criteria changes (39). Inspection of these criteria suggests that our patients with DSM-III schizophrenia who would not meet the DSM-III-R criteria would be in the DSM-III-R category of schizoaffective disorder.

We also used DSM-III criteria for affective disorder, but we restricted unipolar disorder to melancholia. Other forms of depression were not represented in this proband group (mean illness duration=21.91 years, SD=13.34). The DSM-III and DSM-III-R criteria for affective disorder are similar, and our patients with DSM-III affective disorder should also satisfy the DSM-III-R criteria. Our patients with affective disorder had no emotional blunting or avolition and no prolonged psychotic features. Their interepisode functioning was generally good.

We assessed 166 patients with DSM-III schizophrenia and 71 patients with affective disorder. Of the 166 patients with DSM-III schizophrenia, 59 had core schizophrenia, 31 had noncore schizophrenia, and 76 had schizoaffective disorder.

Comparison Probands

We screened prospective comparison probands from the hospital's acute medical wards. Those meeting the screening criteria were assessed by a trained nurse interviewer using a semistructured instrument that is based on the SADS and SADS-L (35) and Present State Examination (40) and includes a detailed psychosocial narrative, which has been suggested as a critical diagnostic factor missing in exclusively structured interviews (41). Our instrument provides the same diagnoses as the SADS and SADS-L ($\kappa=0.92$). In addition, the prospective comparison probands were assessed for avoidant, borderline, schizoid, and schizotypal personality disorders by using operationalized DSM-III criteria checklists. All of this information was then reviewed by two psychiatrists, and only subjects without evidence of past or present psychiatric disorder were included as normal comparison probands ($N=85$).

The comparison probands were about a decade older than the psychiatric probands. Their age (mean=63.14 years, SD=10.50) places them at the upper end of the risk period for psychiatric illness and therefore increases their likelihood of being psychiatrically normal. There were also significantly more Euro-Americans among the comparison subjects than among the psychiatric probands (92% versus 78%; $\chi^2=6.53$, $df=1$, $p<0.05$). However, there is no evidence that Euro-Americans have lower rates of major psychoses than do African-Americans or that there are racial differences in familial transmission of psychosis. The comparison probands also had larger families than did the psychiatric probands, specifically, more children (mean=2.65, SD=2.00). The probands with affective disorder had more children (mean=0.97, SD=1.58) than the schizophrenic probands (mean=0.37, SD=0.93). These differences were not significant. We did not ad-

just the morbid risk figures for this lower fertility rate because of the relatively small numbers of relatives. Such an adjustment generally increases differences between relatives of patients and comparison subjects (42).

Relatives

Initial information about relatives was obtained from the proband and his medical record or social worker. We tried to recruit all first-degree relatives living in the continental United States who could be located by letter and follow-up telephone call.

The relatives were assessed by interviewers using several instruments: the same semistructured interview used to assess the comparison probands, the Schedule for the Assessment of Negative Symptoms and the Schedule for the Assessment of Positive Symptoms, the emotional blunting scale, the Family History Research Diagnostic Criteria (FH-RDC) (43), and our schedules for personality disorders. The interviewers, who were blind to the probands' diagnoses, received extensive training in eliciting, identifying, and recording psychopathology and in the use of all instruments. Quarterly group practice sessions maintained reliability, and the intraclass correlation for each item on each instrument was at least 0.90.

The relatives were diagnosed by two psychiatrists who used information packets that included the aforementioned ratings, FH-RDC information from one to three other first-degree relatives, and available hospital records; the packets did not contain information on the relative's generation or proband affiliation. In addition, periodically information from a proband was transcribed onto the instruments used to assess the relatives, and these pseudorelatives were added to the relative packets. Although there were more relatives than pseudorelatives, the psychiatrists could not be certain whether they were diagnosing a relative or rediagnosing a masked proband. In each case, the masked proband received the same diagnosis that he received when he entered the study.

The diagnostic criteria for psychoses were the same as those used for the probands. We separated unipolar melancholia from other "affective spectrum" depressions (dysthymia; atypical depression; depression not otherwise specified; nonmelancholic major depression; and adjustment disorder, depressed type). We also combined unipolar melancholia and bipolar disorder because the study group was too small to examine these separately and because some forms of unipolar melancholia and bipolar disorder appear to share the same liability (21, 22, 44). We used DSM-III criteria to diagnose all other disorders. A consensus diagnosis was required, and disagreements were resolved by discussion (approximately 10% of the cases).

We identified 1,895 first-degree relatives, of whom 132 (7.0%) were not located and 124 (6.5%) were dead and without mental health information. For an additional 500 (26.4%) relatives who were dead, we had mental health information from one to three FH-RDCs,

the proband's chart, and treaters; this information was sufficient to make blind psychiatric diagnoses for 426 dead relatives. Of the remaining 1,139 living and locatable relatives, 799 (70.1%) participated. Offspring under age 11 ($N=5$) were not interviewed. Of the 523 directly interviewed and blindly diagnosed relatives, 89 (17.0%) were interviewed by telephone. The proportion of telephone interviews ranged from 8% for the relatives of the probands with core schizophrenia to 22% for the relatives of the comparison subjects, but these differences were not statistically significant ($\chi^2=7.61$, $df=4$). These figures for telephone interviews are comparable to those in other family studies (38%) (45). The different proband groups also did not differ significantly with respect to percentage of relatives who were dead with no information (5.3%–7.9%), refused with no information (9.1%–13.9%), or refused with information (4.5%–7.1%).

Of the 799 participating relatives, we interviewed and blindly diagnosed 523. We also blindly diagnosed 426 of the 500 dead relatives with mental health information. For the present report, we combined the direct interview ($N=523$) and family history ($N=426$) information. The majority of the diagnoses for relatives were made from direct interview information (62.6% for DSM-III schizophrenia, 55.7% for unipolar melancholia and bipolar affective disorder, 80.8% for other affective syndromes).

Age Correction

We defined illness onset as age at first psychiatric hospitalization or biological treatment for a psychiatric disorder. The mean ages at illness onset for our probands were 23.93 years ($SD=6.55$) for the schizophrenic probands and 32.06 years ($SD=12.19$) for the probands with affective disorder, which are consistent with those reported for male psychotic subjects (46). Of the probands with core schizophrenia, nearly 80% became ill before age 25, as did 77.8% of the noncore schizophrenic subjects; 41.4% and 37.5%, respectively, had onsets at 21 years of age or earlier. None became ill for the first time after age 39 (core schizophrenia, range=5–37 years; noncore schizophrenia, range=17–39 years). The figures for age at onset by demidecade are also consistent with those for male schizophrenic subjects in community samples, and these data are available for readers who wish to review them. The ages at illness onset for the probands with schizoaffective and affective disorders were 5–48 and 17–62 years, respectively.

We calculated morbid risks according to the Weinberg abridged method (47), using actual proband ages at illness onset to characterize the at-risk ages for the relatives. For the analyses where this was impossible (e.g., our affective spectrum conditions), we used risk periods suggested in the literature (1, 4, 6). Age correction is a standard procedure that permits comparisons among studies of different groups of relatives. Age correction begins with a consensus risk period (i.e., the age

TABLE 3. Morbid Risks for DSM-III Schizophrenia and Affective Disorder in First-Degree Relatives of Schizophrenic, Affectively Ill, and Normal Probands

Proband Group	DSM-III Diagnoses in Relatives (N=949)								
	Schizophrenia			Unipolar and Bipolar Disorder Combined ^b			Affective Spectrum Depressions ^c		
	BZ ^a	Morbid Risk (%)	SE	BZ ^a	Morbid Risk (%)	SE	BZ ^a	Morbid Risk (%)	SE
Schizophrenia (N=166)	335.0	2.69	0.88	283.5	7.05	1.52	291.0	11.68	1.88
Affective disorder (N=71)	156.0	1.92	1.10	135.0	8.88	2.45	139.0	15.83	3.10
Normal subjects (N=85)	263.5	0.00		239.5	2.51	1.01	244.0	9.02	1.83

^aBezugsziffer=age-corrected number of relatives. For DSM-III schizophrenia we used a risk period of 5 to 48 years; for unipolar and bipolar affective disorder combined, 17 to 62 years; and for affective spectrum, 15 to 60 years.

^bFor unipolar disorder we included only patients with melancholia, and for bipolar disorder we combined bipolar I and II (depression must have been melancholia).

^cAdjustment disorder, depressed type; dysthymia; atypical depression or depression not otherwise specified; nonmelancholic major depression.

range in which the illness is likely to develop) and then assumes 1) that there will be no more new cases among relatives not yet in the risk period or beyond the risk period and 2) that among the non-ill relatives in the risk period, each has a 50-50 chance of becoming ill. The number of relatives actually ill (the prevalence) and the total number of relatives studied are then used to calculate the morbid risk. We compared the morbid risks in the relatives of our different proband groups by using the normal deviate (z) (48) for comparing differences in proportions in independent samples.

RESULTS

Table 3 displays the morbid risks in the relatives. The morbid risks for schizophrenia were higher in the relatives of the schizophrenic and affective disorder probands than in the relatives of the comparison subjects ($z=4.64$, $p<0.001$, and $z=3.00$, $p<0.01$, respectively). Although the difference was not statistically significant, the morbid risk for DSM-III schizophrenia was higher in the relatives of the schizophrenic probands than in the relatives of the affective disorder probands ($z=0.82$).

No relative of a comparison proband was diagnosed as schizophrenic, making it difficult to compare them with the relatives of the psychiatric probands because there is no standard error for a morbid risk of zero. We therefore recalculated the normal deviate by using the estimated base rate for schizophrenia suggested by Abrams and Taylor (5) (morbid risk=0.2%) and the observed base rate from the Iowa 500 study (6) (morbid risk=0.6%). The difference between the morbid risks in the relatives of the schizophrenic and comparison probands remained significant with both base rates (morbid risk of 0.2%: SE=0.28, $z=3.83$, $p<0.01$; morbid risk of 0.6%: SE=0.48, $z=3.03$, $p<0.01$). The difference in morbid risks between the relatives of the affective disorder and comparison probands also remained significant with the 0.2% base rate (SE=0.28, $z=2.23$, $p<0.05$) but fell short of significance with the 0.6% base rate (SE=0.48, $z=1.55$).

The morbid risks for combined unipolar melancholia and bipolar affective disorder were also higher in the relatives of the schizophrenic and affectively ill probands than in the relatives of the comparison subjects ($z=4.59$, $p<0.001$, and $z=4.83$, $p<0.001$, respectively). The risk was highest in the relatives of the probands with affective disorder, but it was not significantly higher than in the relatives of the schizophrenic subjects ($z=1.31$). The morbid risk for affective spectrum disorders in the relatives of the schizophrenic probands was not significantly higher than that of the relatives of the comparison subjects ($z=2.27$), but it was significantly higher in the relatives of the affective disorder probands than in the relatives of the comparison subjects ($z=4.28$, $p=0.01$) or the relatives of the schizophrenic probands ($z=2.69$, $p<0.05$).

Table 4 displays the morbid risks in the relatives of the probands with core schizophrenia, noncore schizophrenia, and schizoaffective disorder. The risks for DSM-III schizophrenia and affective disorder did not differ significantly among the relatives of these subgroups, although the risk for affective disorder was highest among the relatives of the probands with core schizophrenia, and the risk for DSM-III schizophrenia was highest among the relatives of the schizoaffective subgroup. Combining the relatives of the probands with core and noncore schizophrenia into a group with nonaffective psychotic disorder did not alter the morbid risk differences. The morbid risks for affective spectrum disorders did not differ significantly among the groups.

The morbid risks for DSM-III schizophrenia and for combined unipolar melancholia and bipolar disorder in the relatives of each DSM-III schizophrenia proband subgroup were higher than in the relatives of the comparison subjects. The relatives of the probands with core schizophrenia ($z=2.69$, $p<0.05$), noncore schizophrenia ($z=2.08$, $p<0.05$), core and noncore schizophrenia combined ($z=2.87$, $p<0.05$), and schizoaffective disorder ($z=4.85$, $p<0.001$) all had significantly higher risks for DSM-III schizophrenia than did the relatives of the comparison probands. With the base rate of 0.6% (SE=0.48), the morbid risks for DSM-III schizo-

TABLE 4. Morbid Risks for DSM-III Schizophrenia and Affective Disorder in First-Degree Relatives of Subgroups of Probands With DSM-III Schizophrenia

Proband Group	DSM-III Diagnoses in Relatives (N=949)								
	Schizophrenia			Unipolar and Bipolar Disorder Combined ^b			Affective Spectrum Depressions ^c		
	Morbidity Risk (%)			Morbidity Risk (%)			Morbidity Risk (%)		
	BZ ^a	Risk	SE	BZ ^a	Risk	SE	BZ ^a	Risk	SE
Core and noncore schizophrenia (N=90)	165.5	1.81	1.04	148.0	8.11	2.24	150.5	9.97	2.44
Core (N=59)	104.5	1.91	1.34	95.5	8.38	2.84	97.0	9.28	2.95
Noncore (N=31)	61.0	1.64	1.63	52.5	7.62	3.66	53.5	11.21	4.31
Schizoaffective disorder (N=76)	169.5	3.54	1.42	142.5	5.61	1.93	148.0	12.84	2.94

^aBezugsziffer=age-corrected number of relatives. For DSM-III schizophrenia we used a risk period of 5 to 48 years; for unipolar and bipolar affective disorder combined, 17 to 62 years; for affective spectrum, 15 to 60 years.

^bFor unipolar disorder we included only patients with melancholia, and for bipolar disorder we combined bipolar I and II (depression must have been melancholia).

^cAdjustment disorder, depressed type; dysthymia; atypical depression or depression not otherwise specified; nonmelancholic major depression.

phrenia in the relatives of the probands with core schizophrenia ($z=1.32$) or core and noncore schizophrenia combined ($z=1.46$) again fell short of significance. The morbid risk for noncore schizophrenia alone also became nonsignificant ($z=0.88$), but the number of relatives was small. The morbid risk for DSM-III schizophrenia remained significant for the relatives of the schizoaffective probands ($z=3.27$, $p<0.01$). Compared to the relatives of the comparison probands, there were significantly higher risks for unipolar melancholia and bipolar disorder combined in the relatives of the probands with core schizophrenia ($z=3.94$, $p<0.01$), noncore schizophrenia ($z=2.76$, $p<0.05$), core and noncore combined ($z=4.44$, $p<0.001$), and schizoaffective disorder ($z=2.54$, $p<0.05$).

Eight percent of the probands with core schizophrenia and 3% of those with noncore schizophrenia each had an affectively ill family member. In many of these pedigrees several first-degree relatives had affective disorders and others had schizophrenic syndromes. In no family was one parent schizophrenic and the other affectively ill.

DISCUSSION

Our data suggest a familial relationship between DSM-III schizophrenia and affective disorder. They also suggest this relationship may be between schizophrenia and unipolar melancholia/bipolar disorder, because only the risks for these disorders, and not those for affective spectrum disorders, were higher than normal in the relatives of the probands with DSM-III schizophrenia. Such a relationship was suggested by Gershon et al. (4, 9) and is consistent with the findings by Kendler et al. (14) of a higher than expected risk for schizophrenia (4.3%) in the relatives of probands with "psychotic affective disorder" but not in relatives of patients with nonpsychotic forms. This may explain some of the apparent failures to find familial co-occurrence

in previous studies, in which the base rates for affective disorder used to compare groups were quite high (about 6%) (49), resulting from inclusion of nonmelancholic major depression and dysthymia as part of manic-depressive illness. Earlier European data (50) suggest a risk of 1%, as do lithium treatment data from Canada (51) and Swedish population rates of 6.9% for all major depression but only 2.9% for melancholia (52). Use of this low figure would result in higher risks for affective disorder in the first-degree relatives of schizophrenic subjects in several studies with negative findings. As depression may be heterogeneous (53), it is important to determine which variety co-occurs in relatives of some schizophrenic subjects.

Kendler and Hays (54) suggested that schizophrenic subjects with family histories of affective disorder may be affectively ill, and about 45% of our patients with DSM-III schizophrenia met the criteria for schizoaffective disorder. However, our core and noncore subtypes were based on the absence of affective episodes, and the relatives of these subgroups had high risks for affective disorder that were similar to the risk for the relatives of our affectively ill probands. Further, when compared with the core and noncore schizophrenic probands without family histories of affective disorder, those with such family histories had an earlier mean onset (20.7 versus 23.2 years), a higher mean summed negative symptom score (44.67 versus 35.50), a similar nonaffective positive symptom score (18.04 versus 17.95), and, most important, lower scores for depressive (0.33 versus 3.20) and manic (0.00 versus 0.32) features. Core and noncore schizophrenia are markedly different from the classic concept of affective disorder, but they are similar to the classic dementia praecox. It seems unlikely that these patients are affectively ill patients masquerading as schizophrenic and, thus, producing spurious co-occurrence.

At first glance, the morbid risk of 2.69% for DSM-III schizophrenia in the relatives of our DSM-III schizophrenic probands seems low. However, in several re-

views (2, 25, 34) of family studies done since 1980 in which DSM-III or specific research criteria were used, the weighted average morbid risk was about 5%, and Kendler (2) reported a risk of 3.7%. Further, there is evidence (55–57) that compared to the relatives of female schizophrenic patients, the relatives of male schizophrenic patients have a lower morbid risk for schizophrenia. This has been attributed (57) to more phenocopies (and, thus, less genetic loading) among male patients. Our risk finding is consistent with this literature.

Our analyses are limited. First, we combined family history and interview information. However, it is unlikely that this explains our results, as the use of direct interview and family history information did not differ among the relative groups. Second, the study group was too small for full analysis of the differences among the schizophrenic subgroups and for determination of whether the apparent relationship between affective disorder and schizophrenia pertains to both unipolar and bipolar disorder. Third, the zero base rate for DSM-III schizophrenia in the relatives of the comparison probands made it difficult to compare morbid risks. This lack of observed DSM-III schizophrenia in these relatives may reflect our selection of normal comparison subjects. Fourth, to demonstrate a shared liability, two relationships need to be established: a higher than normal risk of affective disorder in relatives of schizophrenic probands and a higher than normal risk of schizophrenia in relatives of affective disorder probands. Our data indicate the former but only suggest the latter.

Thus, our data need to be expanded. Nevertheless, in addition to the studies previously mentioned, they suggest that the Kraepelinian view of psychosis may need modification. Further research should focus on factors that may account for differences across studies, e.g., number of subjects, illness severity of the schizophrenic and affective disorder probands, severity and type of depression, and the overlap between unipolar and bipolar disorders. Additional study should also focus on nongenetic factors (street drugs, perinatal problems, viral disease) that might alter the clinical expression of a shared psychosis-prone genotype.

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Pathological Laughing and Crying Following Stroke: Validation of a Measurement Scale and a Double-Blind Treatment Study

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Objective: This study was undertaken to test the reliability and validity of the Pathological Laughter and Crying Scale and the effectiveness of nortriptyline treatment for patients with emotional lability following stroke. **Method:** Eighty-two patients with ischemic brain injury—54 who had been hospitalized with acute stroke and 28 others who requested treatment for pathological laughing and crying—were given standardized psychiatric and neurological assessments and then administered the Pathological Laughter and Crying Scale. The 54 acute stroke patients were used to evaluate the Pathological Laughter and Crying Scale, and the 28 patients with pathological emotional display were randomly assigned to nortriptyline treatment or placebo in a 6-week double-blind trial to assess the efficacy of a tricyclic antidepressant in treatment of this disorder. **Results:** The interrater reliability on the Pathological Laughter and Crying Scale for a subgroup of 15 patients was 0.93, and the test-retest reliability of the scale was excellent. After 4 and 6 weeks of treatment, scores on the Pathological Laughter and Crying Scale showed significantly greater improvement in the 14 patients given nortriptyline than in the 14 given placebo. Although almost one-half of these patients also had major depression, the improvement in emotional lability was independent of depression status. In addition, response to treatment was not significantly affected by lesion location or time since stroke. **Conclusions:** The severity of symptoms in pathological emotional display can be reliably quantified with the Pathological Laughter and Crying Scale, and treatment with nortriptyline can effectively ameliorate this emotional disorder.

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Frequent episodes of crying or laughing without feelings of sadness or happiness (sometimes referred to as emotional lability, pseudobulbar affect, emotional incontinence, or pathological crying and laughing) have been recognized by clinicians since the late nineteenth century as a common manifestation of brain damage (1–3). Some clinicians have distinguished

between the clinical presentation of emotional lability, in which there is easy vacillation from laughter to crying, and the presentation of pathological or pseudobulbar crying or laughing, frequently associated with paralysis of the voluntary facial muscles and bilateral or diffuse brain injury (4). The overlap, however, between these various forms of emotional lability is marked, and it has never been established whether they have the same or different mechanisms or etiologies (5).

There is general agreement that a diagnosis of pathological laughter or crying should be based on the occurrence of frequent or intense episodes of laughter or crying which are in excess of an “appropriate” emotional response to the provoking stimulus (6). There has not, however, been a consensus about the magnitude of this dissociation needed for making the diagnosis. Sometimes patients may be depressed, but the frequency and/or intensity of their crying episodes seem to be excessive for their underlying mood state. In other cases, however, the episodes of crying seem to occur with no provocation. In addition to this uncertainty about the criteria necessary for the diagnosis of pathological laughter and crying, there are no established rating

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scales that can be used to quantify the degree of emotional lability. Previous investigators (7, 8) have made clinical judgments about the degree of emotional lability and the changes associated with treatment, but no scale with established reliability and validity has been used in these studies.

In addition to this problem of clinical definition and measurement of severity, there is the issue of need for treatment. The socially debilitating effects of crying or laughing inappropriately have been described by numerous authors (8, 9). Fears of developing an uncontrollable emotional display can lead to social phobia and withdrawal. Previous investigators have reported, in both anecdotal and double-blind controlled studies of emotional lability associated with brain lesions, that tricyclic antidepressants are effective in the treatment of these disorders (8, 9). There has never been, however, a double-blind trial of treatment for emotional lability among patients with stroke.

In the present study, we tried to address these issues. This report concerns the validation of the Pathological Laughter and Crying Scale and the effectiveness of nortriptyline in the treatment of emotional lability following stroke.

METHOD

The study included two groups of patients. The first consisted of 54 consecutive patients admitted to a university hospital with acute thromboembolic or intracerebral hemorrhagic infarction. This group was used to assess the reliability and validity of the Pathological Laughter and Crying Scale. All of these patients were included in the Stroke Data Bank of the National Institute of Neurological Disorders and Stroke (10). We have described this Stroke Data Bank population in previous reports (11). Patients with decreased levels of consciousness or moderate to severe aphasia with deficits in comprehension (approximately one-fourth of patients admitted because of an acute stroke) were excluded. The second group consisted of 28 additional patients (there was no overlap between the patients in groups 1 and 2) without deficits in comprehension who were participants in our double-blind treatment study. Patients in the treatment study either were self-referred in response to newspaper advertisements or word-of-mouth announcement of our study or were recommended by their physicians for treatment of pathological emotions or depression with excessive crying. There was no other bias, and all patients referred were entered in the study (three patients dropped out). The majority ($N=22$) of these patients were seen as outpatients, although six participants were inpatients. The pretreatment evaluations of these study patients were also used for the reliability and validity studies. Informed consent for participation in this study was obtained from all patients.

Neurological evaluations of the hospitalized patients were done by the attending neurologist using the stand-

ardized examination and rating criteria from the Stroke Data Bank (10), and those of the outpatients were done by the examining neurologist following clinical assessment. For both inpatients and outpatients, CT scans were performed at the treating hospital during their admission for acute stroke. Psychiatric examination included a structured psychiatric interview, the Present State Examination (PSE) (12), which was used to establish DSM-III-based diagnoses of mood disorder. In addition, the examiner administered the 18-item Hamilton Rating Scale for Depression (13) to quantify the severity of depressive symptoms. Both instruments have been demonstrated to be reliable and valid for patients with stroke (14). We have modified the PSE to rate primarily symptoms of anxiety and mood (14). In order to minimize any possible effect of diurnal mood variation, patients' interviews were conducted in private during the late morning or early afternoon.

Activities of daily living were measured with the 10-item Johns Hopkins Functioning Inventory (15). Scores may range from 0 to 27, with higher scores indicating greater impairment. Cognitive impairment was assessed with the Mini-Mental State examination (16), which evaluates intellectual functions, particularly those related to language. Scores range from 0 to 30, with scores below 23 indicating significant cognitive impairment. The number of social connections was ascertained with the Social Ties Checklist (17). Scores range from 0 to 10, with higher scores indicating poorer social functioning.

All 82 patients were administered the Pathological Laughter and Crying Scale (appendix 1). This is an interviewer-rated instrument that quantifies aspects of laughter and crying, including the relation of the episodes to external events, duration, degree of voluntary control, inappropriateness in relation to emotions, and degree of resultant distress. The scale was administered to the patients and to relatives or friends who had been in close contact with the patients for the preceding 2 weeks. For each of the items, the examiner made a judgment about the severity of the symptom on a scale of 0–3 points. Scores on all items were totaled to obtain an overall score. Eight items relate to pathological laughter and eight to pathological crying. To determine the validity of the Pathological Laughter and Crying Scale score, diagnoses of emotional lability were made by examiners blind to the patients' scale scores who used their clinical judgment (e.g., was emotional lability displayed during the interview? did it occur several times daily? did it seem excessive in relation to the precipitant?). A subgroup of 15 patients were randomly selected and administered the Pathological Laughter and Crying Scale independently by two psychiatrists in order to evaluate the interrater reliability of the instrument.

The 28 patients participating in the treatment study were given nortriptyline or placebo (in identical capsules) in a single daily dose at bedtime after random-number assignment to either active treatment or placebo. Both the patients and the examiners were unaware of which treatment was being given. Patients

TABLE 1. Characteristics of 82 Stroke Patients in a Study of Pathological Laughing and Crying

Characteristic	Patients Given Nortriptyline in Treatment Study (N=14)	Patients Given Placebo in Treatment Study (N=14)	Patients in Reliability-Validity Study of Rating Scale ^a (N=54)
Female sex	6	10	25
White race ^b	7	12	19
Married	7	10	24
Socioeconomic class IV or V	7	7	36
Prior cardiovascular accident	7	4	8
Right-handed	10	14	48

^aPathological Laughter and Crying Scale.^bSignificant difference between groups ($\chi^2=11.5$, $df=2$, $p<0.004$).

were given 20 mg/day for 1 week, 50 mg for 2 weeks, 70 mg for 1 week, and 100 mg for the last 2 weeks of the study. They were evaluated before treatment began and at 2-week intervals during the 6-week treatment period. The neurological examination and complete psychiatric examination were performed at the beginning of the treatment study. The Pathological Laughter and Crying Scale, Hamilton depression scale, Mini-Mental State examination, and Johns Hopkins Functioning Inventory were given at each follow-up evaluation, and serum was drawn for determination of nortriptyline concentrations by a standardized gas chromatography method (18).

The CT scans were evaluated by a neurologist (S.E.S.) who was experienced in the assessment of CT images but was unaware of any of the clinical findings. The location of the lesion was determined by assessing which structures were involved. Lesion volume was determined by measuring the maximal area within the lesion as determined by a computer-assisted area calculation program and then dividing by the overall brain area in the slice which passes through the body of the lateral ventricles. These methods were detailed in a previous publication (19).

Means and standard deviations were computed for all parametric data. Intraclass and Pearson correlation coefficients were calculated for all data obtained from interval scale measures. Intergroup comparisons were made by using repeated measures analyses of variance (ANOVA) with planned comparison *t* tests (two-tailed) for individual comparisons. Nonparametric between-group comparisons were made with chi-square tests.

RESULTS

Reliability-Validity Study

The background characteristics of the 54 patients included in the study of the reliability and validity of the Pathological Laughter and Crying Scale are shown in table 1. The patients were predominantly elderly (mean

age=60.5 years, $SD=16.2$) and black and were from lower socioeconomic classes. They were evaluated within the first few months after acute cerebral infarction (mean=1.6 months, $SD=2.5$).

The intraclass correlation coefficient for the assessments made by the two psychiatrists who separately administered the Pathological Laughter and Crying Scale to 15 patients was 0.93 ($p<0.01$). The Pearson correlation coefficient for test-retest reliability of this instrument when administered at 2-week intervals was 0.85 ($N=21$, $p<0.01$).

The validity of the Pathological Laughter and Crying Scale was assessed first by comparing the mean score of 17 patients (including some in the treatment group) who had a clinical diagnosis of emotional lability made by a psychiatrist who was blind to the patients' scale scores and the mean score of 50 patients without emotional lability. An ANOVA revealed significantly higher scores in the emotional lability group (mean=16.2, $SD=3.1$) than in the no-lability group (mean=2.7, $SD=4.8$) ($F=144.9$, $df=1$, 65, $p<0.001$). Using a score of 13 or greater (arbitrarily selected because it seemed to distinguish those with and without lability), we found that the sensitivity of the Pathological Laughter and Crying Scale for clinically diagnosed emotional lability was 0.88 ($N=67$). The specificity was 0.96, and the positive predictive value was 0.83. Patients' scores on this scale were also correlated with scores obtained by interviewing their relatives or other persons with whom they had close relationships. The Pearson correlation coefficient was 0.86 ($N=28$) between scores obtained from interviews with patients and those from interviews with relatives. Finally, there were no significant correlations between Pathological Laughter and Crying Scale score and Hamilton depression score ($r=0.09$), Mini-Mental State score ($r=0.01$), Johns Hopkins Functioning Inventory score ($r=0.05$), or Social Ties Checklist score ($r=0.09$) ($N=28$ for each correlation). These findings indicate that the Pathological Laughter and Crying Scale was assessing a factor other than the ones being measured by these instruments.

Treatment Study

Characteristics of the patients. The background characteristics of the 28 patients included in the treatment study are shown in table 1. There was only one patient who dropped out during the course of the study (not included among the 28 study completers). This was a 78-year-old man who was receiving active medication but dropped out between weeks 2 and 4 because of complaints of sedation. An additional two patients dropped out, on the advice of their family physicians, after the evaluation but before taking any medication. There were no adverse side effects that necessitated withdrawal from the study in any of the remaining patients, all of whom completed the 6-week protocol. There were no statistically significant differences between the nortriptyline and placebo groups in age (mean=57.8 years, $SD=10.1$, and mean=58.5 years,

SD=11.8, respectively), sex distribution, race, socioeconomic status, marital status, previous cardiovascular accident, handedness, or time since stroke (mean=8.1 months, SD=9.9, and mean=15.7 months, SD=13.5, respectively).

Neurological and CT scan findings. There were no significant differences between the two groups in the treatment study in the nature of the strokes and the frequency of hemiparesis or monoparesis, sensory deficit, visual field disturbance, or aphasia (table 2). On the basis of CT scans or clinical findings for lesion location, 10 of 26 patients (lesion location could not be determined in two patients) had single right hemisphere lesions, and eight of the 26 had bilateral or multiple lesions. There were, however, no significant differences between the nortriptyline and placebo groups in the frequency of right, left, bilateral, cortical, or subcortical lesion locations (table 2) or lesion volume (mean for the nortriptyline group=9.1% of brain volume, SD=6.4%; mean for the placebo group=8.0%, SD=7.8%).

Psychiatric evaluation. The pretreatment evaluation of study patients revealed no significant differences between the active drug and placebo groups in scores on the Mini-Mental State examination, the Johns Hopkins Functioning Inventory (activities of daily living), or the Social Ties Checklist (table 3). There was, however, a significantly higher mean pretreatment Hamilton depression score for the placebo group than for the nortriptyline group. With the diagnosis of depression based on DSM-III criteria (excluding the no-organic-factors criterion) and symptoms elicited by the PSE, eight of the nortriptyline-treated patients and 11 of the patients who received placebo had major depression. Repeated measures ANOVA of pretreatment versus posttreatment scores revealed a significant decline (improvement) in scores on the PSE and the Hamilton depression scale in both the active drug group and the placebo group, but a significantly greater improvement in the group treated with the active drug (table 3). There was a trend for the Mini-Mental State scores to improve in the group given nortriptyline but not in the placebo group (table 3).

Pathological Laughter and Crying Scale findings. Repeated measures ANOVA of the Pathological Laughter and Crying Scale scores demonstrated a significant group effect ($F=8.3$, $df=1, 26$, $p=0.008$) (i.e., the group treated with nortriptyline showed significantly greater overall improvement than the placebo group), a significant time effect ($F=42.2$, $df=3, 78$, $p<0.001$) (i.e., both groups improved over time), and a significant Group by Time interaction ($F=10.2$, $df=3, 78$, $p<0.001$) (i.e., the group treated with the active drug improved more quickly than the placebo group). Planned comparisons revealed that the active drug and placebo groups were not significantly different in their Pathological Laughter and Crying Scale scores at the beginning of the study and at week 2 (mean=9.6, SD=4.0, and mean=11.4, SD=5.7, respectively) but that the nortriptyline group was significantly more improved than the placebo group at week 4 (mean=4.1,

TABLE 2. Neurological Findings in 28 Stroke Patients With Pathological Laughing or Crying Given a 6-Week Double-Blind Trial of Nortriptyline or Placebo

Finding	Patients Given Nortriptyline (N=13) ^a	Patients Given Placebo (N=13) ^a
Type of stroke		
Thromboembolic	12	10
Hemorrhagic	1	3
Lesion location		
Right hemisphere (single lesion)	4	6
Left hemisphere (single lesion)	2	3
Bilateral/multiple	5	3
Brainstem	2	1
Cortical	4	6
Subcortical	5	3
Mixed (cortical plus subcortical)	3	1
Neurological problem		
Monoparesis/hemiparesis	7	11
Sensory deficit	1	4
Visual field deficit	3	0
Aphasia	2	5

^aData were not available for one patient in this group.

SD=3.8, and mean=10.6, SD=4.7, respectively; $t=3.98$, $df=26$, $p=0.005$) and at week 6 (mean=1.2, SD=2.0, and mean=9.6, SD=5.7, respectively; $t=5.14$, $df=26$, $p=0.0001$) (figure 1).

Because the nortriptyline-treated group had significantly lower Hamilton depression scores than the placebo group, we matched eight pairs of patients in the nortriptyline and placebo groups on Hamilton scores (± 4 points). Although their pretreatment Hamilton scores were not significantly different (mean=15.9, SD=4.2, for the nortriptyline group, and mean=16.5, SD=5.3, for the placebo group), the nortriptyline group showed a significantly greater improvement in Pathological Laughter and Crying Scale scores than the placebo group ($F=13.7$, $df=1, 14$, $p=0.002$), a significant repeated measures effect ($F=20.1$, $df=3, 42$, $p=0.001$), and a significant interaction ($F=20.3$, $df=3, 42$, $p=0.001$).

We next examined the effect of a diagnosis of major depression on response to treatment. Patients with depression and pathological laughing or crying ($N=8$ in the nortriptyline group, and $N=11$ in the placebo group) were compared with patients without depression ($N=6$ and $N=3$, respectively) for change in Pathological Laughter and Crying Scale scores with active treatment and placebo. A two-way repeated measures ANOVA of the scores of the depressed and nondepressed groups revealed a highly significant effect of active treatment versus placebo over time ($F=14.4$, $df=3, 72$, $p<0.001$) but no effect of depression ($F=1.8$, $df=3, 72$, n.s.) (i.e., depressed and nondepressed patients responded equally). There was a trend toward a significant interaction ($F=2.7$, $df=3, 72$, $p<0.06$) (i.e., the nondepressed patients tended to improve more in this scale score than the depressed group).

We also investigated the effect of lesion location on response to treatment. Patients with single right hemi-

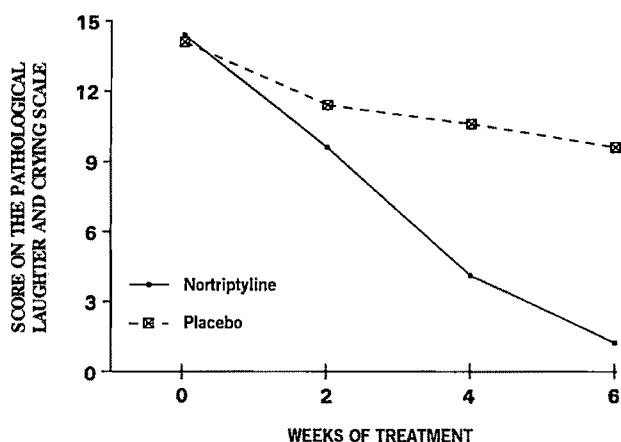
TABLE 3. Scores on Psychological Tests Before and After a 6-Week Double-Blind Trial of Nortriptyline or Placebo for 28 Stroke Patients With Pathological Laughing or Crying

Test	Patients Given Nortriptyline (N=14)				Patients Given Placebo (N=14)			
	Score Before Treatment		Score After Treatment		Score Before Treatment		Score After Treatment	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Present State Examination ^a	17.5	10.0	1.8	2.3	28.8	8.5	16.0	9.8
Hamilton Rating Scale for Depression ^b	12.1	7.3	2.1	2.4	18.6	5.2	11.3	7.9
Mini-Mental State ^c	24.1	5.1	26.5	4.8	26.5	3.5	26.8	3.0
Johns Hopkins Functioning Inventory	4.7	6.0	3.7	5.0	6.0	5.9	5.1	5.3
Social Ties Checklist	4.0	1.6	3.6	1.4	4.2	1.9	4.2	1.9

^aSignificant difference between pretreatment and posttreatment scores within both groups (nortriptyline, paired $t=5.9$, $df=12$, $p<0.0001$; placebo, $t=3.4$, $df=12$, $p<0.01$), but significantly greater improvement in the nortriptyline group ($F=27.6$, $df=1, 25$, $p<0.001$).

^bSignificantly higher pretreatment score in the placebo group ($t=2.6$, $df=26$, $p<0.02$). Significant difference between pretreatment and posttreatment scores within both groups (nortriptyline, paired $t=5.5$, $df=13$, $p<0.0001$; placebo, $t=2.8$, $df=13$, $p<0.01$), but significantly greater improvement in the nortriptyline group ($F=17.5$, $df=1, 26$, $p<0.001$).

^cTrend toward significant difference between pretreatment and posttreatment scores within the nortriptyline group ($F=2.9$, $df=1, 26$, $p=0.10$).

FIGURE 1. Mean Scores on the Pathological Laughing and Crying Scale of 28 Stroke Patients During a 6-Week Double-Blind Trial of Nortriptyline Treatment or Placebo

sphere lesions (N=4 in the nortriptyline group, and N=6 in the placebo group) were compared with patients having bilateral lesions (N=4 and N=3, respectively). Two-way repeated measures ANOVA revealed a significant effect of active drug treatment versus placebo ($F=9.7$, $df=3, 42$, $p=0.0001$) but no effect of lesion location on response to treatment ($F=2.7$, $df=3, 42$, $p=0.06$) (i.e., the right hemisphere and bilateral groups improved to the same degree with nortriptyline) and no significant interaction ($F=0.4$, $df=3, 42$, $p=0.73$).

Finally, we examined the effect of time since stroke on treatment response. Because the nortriptyline patients were evaluated an average of 8.1 months after stroke and the placebo patients an average of 15.7 months after stroke (a statistically nonsignificant difference, however), we wanted to be sure that the placebo patients were not a more chronic, treatment-resistant group than the nortriptyline patients. We matched eight pairs of patients for time since stroke (i.e., the exact number of months up to 12 months, within ± 3 months up to 24 months, and within ± 6 months for

more than 24 months); the mean for the nortriptyline group was 10.9 months, $SD=11.2$; the mean for the placebo group was 11.3 months, $SD=10.3$). Repeated measures ANOVA revealed a significant time interaction effect of active versus placebo treatment ($F=8.5$, $df=3, 42$, $p<0.001$). Thus, group differences in time since stroke did not explain our findings that nortriptyline significantly improved emotional lability scores.

Most of the patients had pathological crying, but two patients had pathological laughing. The one patient with laughter who was given nortriptyline responded (the Pathological Laughing and Crying Scale score was 27 before treatment and 0 at week 6). The placebo-treated patient did not respond (scores were 32 at week 0, 31 at week 2, 31 at week 4, and 27 at week 6).

Serum levels of nortriptyline. Mean serum levels of nortriptyline in the active treatment group were 66.7 ng/ml ($SD=46.1$) at week 2 (following an oral nortriptyline dose of 50 mg/day for 1 week and 12–18 hours after the last dose), 72.0 ng/ml ($SD=26.3$) at week 4 (oral dose=70 mg/day for 1 week), and 121.6 ng/ml ($SD=62.8$) at week 6 (oral dose=100 mg/day for 2 weeks).

DISCUSSION

This study demonstrated that emotional lability following stroke can be effectively treated with the tricyclic antidepressant nortriptyline. In addition, we have developed a scale that is both reliable and valid for the quantification of emotional lability and that showed its usefulness in measuring outcome in this double-blind treatment study.

Before we discuss these findings further, the limitations of the study should be acknowledged. Patients included in the treatment study were not identified from a consecutive series of patients hospitalized for stroke or attending an outpatient clinic but were outpatients requesting treatment for emotional lability or patients hospitalized for stroke rehabilitation who were willing to undergo treatment. Whether the results in this group

would be comparable to those in a consecutive series of stroke patients with emotional lability is unknown. Some of the patients had depression as well as emotional lability, and the mean time between stroke and evaluation for the study for the entire study group was 11.9 months. Brain lesions were assessed with CT scans, which may have failed to visualize some ischemic lesions. Thus, our failure to find strong anatomical correlates of emotional lability (e.g., multiple lesions) may have been due to our neuroimaging techniques. We also did not have enough patients to draw any conclusions about whether pathological laughter is more or less likely to respond to treatment than pathological crying. Thus, the findings from this study should be examined in other groups of patients with emotional lability following stroke.

In spite of these limitations, however, this study demonstrated, for the first time, with the use of double-blind controls, that emotional lability occurring after stroke can be effectively treated with nortriptyline.

Since more than one-half of our study patients met the diagnostic criteria for a depressive disorder, one might reasonably question whether we were simply treating depressed patients and the response to treatment was related to depression, not pathological laughing and crying. Ross and Stewart (20) suggested that in some patients, the combination of depression and a right hemisphere lesion involving neocortical control of limbic-associated motor behaviors is necessary to produce pathological laughing and crying. These authors also suggested that pathological affect in these patients is indicative of the presence of depression. There are several lines of evidence from our study, however, which suggest that depression and pathological crying may be independent disorders. First, scores on the Pathological Laughter and Crying Scale did not show an effect of depression in two-way ANOVAs or a significant correlation with Hamilton depression scores. This suggests that depression and emotional lability are distinct disorders, even though they coexist in some patients. Second, when we examined patients with emotional lability but without depression, we found that these patients did as well when treated with nortriptyline as patients with both depression and emotional lability. Schiffer et al. (8), in their study of pathological laughing and crying in patients with multiple sclerosis, also concluded that depression and emotional lability were separate disorders, both of which respond to antidepressants.

The mechanism by which antidepressants ameliorate pathological laughing and crying remains unknown. The traditional explanation for pathological laughing and crying (pseudobulbar affect) is that bilateral interruption of neocortical upper motor neuron innervation of bulbar motor nuclei leads to this symptom by an undetermined mechanism involving loss of descending fibers to the brainstem (21). The fact that antidepressants have been shown to be effective in treating emotional lability associated with other disorders such as multiple sclerosis (8) suggests that the mechanism underlying emotional lability may be similar in a variety of neuropathological conditions and that the disorder may in-

volve dysfunction of biogenic amine systems in the brain. We have shown that the amount of serotonin 5_2 receptor binding in the left temporal cortex was significantly correlated with the severity of depressive symptoms following stroke (22). Thus, one hypothesis to explain the present findings is that dysfunction of the serotonergic pathways might destabilize the input from the basotemporal limbic cortex to the amygdala and lateral limbic circuit, leading to brief outbursts of crying or laughing without the usual cortical cognitive associations. Although other explanations might also be proposed, these findings do suggest that the mechanism of pathological laughing and crying is probably not the result of a focal abnormality but, rather, a pathophysiological process which can arise from a wide variety of diffuse or focal lesions and may be mediated by a dysfunction of the biogenic amine systems.

There are several implications of these study findings. First, pathological laughing or crying can be quantitatively assessed, and therefore patients can be systematically evaluated for severity of this disorder. Second, although patients may vary in the severity of their emotional lability, even patients with mild cases may be able to be helped considerably by treatment with an antidepressant, whether or not there is accompanying depression. Thus, patients who have this disorder following stroke should be evaluated and treated if there is no medical contraindication for the administration of nortriptyline. Patients with coexisting disorders such as severe cardiac arrhythmia, conduction delay, prostatic urinary obstruction, orthostatic hypotension, or narrow angle glaucoma must be treated cautiously, with frequent medical monitoring. Whether other antidepressants or psychotropic medications would be just as effective as nortriptyline in treating the disorder is an issue that awaits further controlled treatment trials. Ultimately, the goal of developing truly rational (as opposed to empirical) treatments, however, will require the identification of pathophysiological mechanisms leading to this disorder.

In summary, this study has shown that pathological laughing and crying after stroke can be quantified and can be effectively treated with the tricyclic antidepressant nortriptyline. Further empirical trials examining other groups of patients with stroke, as well as treatment with other medications, need to be conducted. Although the mechanisms underlying this fascinating but socially devastating disorder remain unknown, the findings from this study as well as others suggest that subcortical mechanisms, including dysfunction of the biogenic amine input to the limbic pathways, may lead to a display of mood symptoms without congruent cognitive processes.

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APPENDIX 1. Pathological Laughter and Crying Scale (Patient Interview)

Ratings are based on clinical assessment. Initial probe questions are given for each item. However, further questions may be used for clarification. Write the number in the spaces provided which most accurately reflects clinical symptoms.

1. Have you recently experienced sudden episodes of laughter?
 ____ Rate the frequency of the episodes during the past two weeks.
 0. Rarely or not at all
 1. Occasionally
 2. Quite often
 3. Frequently
2. Have you recently experienced sudden episodes of crying?
 ____ Rate the frequency of the episodes during the past two weeks.
 0. Rarely or not at all
 1. Occasionally
 2. Quite often
 3. Frequently

If you have experienced sudden episodes of laughter, please answer the following (questions 3-10), otherwise skip to question 11.

3. Have these episodes occurred without any cause in your surroundings?
 ____ Rate the frequency with which the episodes have occurred without external stimuli in the past two weeks.
 0. Rarely or not at all
 1. Occasionally
 2. Quite often
 3. Frequently
4. Have these episodes lasted for a long period of time?
 ____ Rate the average duration of the episodes during the past two weeks.

0. Very brief
 1. A few seconds
 2. Moderate (less than 30 seconds)
 3. Prolonged (more than 30 seconds)
5. Have these episodes been uncontrollable by you?
 ____ Rate the ability to control the episodes during the past two weeks.
 0. Rarely or not at all
 1. Occasionally
 2. Quite often
 3. Frequently
6. Have these episodes occurred as a result of feelings of happiness?
 ____ Rate the frequency with which the episodes have occurred as a result of happiness in the past two weeks.
 0. Rarely or not at all
 1. Occasionally
 2. Quite often
 3. Frequently
7. Have these episodes occurred in excess of feelings of happiness?
 ____ Rate the frequency with which the episodes have been disproportionate to the emotional state in the past two weeks.
 0. Rarely or not at all
 1. Occasionally
 2. Quite often
 3. Frequently
8. Have these episodes of laughter occurred with feelings of sadness?
 ____ Rate the frequency of association between the episode and the paradoxical emotion in the past two weeks. The sadness must precede or accompany the episode and not be a reaction to it.

- 0. Rarely or not at all
 - 1. Occasionally
 - 2. Quite often
 - 3. Frequently
9. Have these episodes occurred with any emotions other than happiness or sadness, such as nervousness, anger, fear, etc.?
- ___ Rate the frequency of association between the episodes and emotions in the past two weeks. The emotions must precede or accompany the episode and not be a reaction to it.
- 0. Rarely or not at all
 - 1. Occasionally
 - 2. Quite often
 - 3. Frequently
10. Have these episodes caused you any distress or social embarrassment?
- ___ Rate the degree of distress or embarrassment caused by the episodes in the past two weeks
- 0. Rarely or not at all
 - 1. Occasionally
 - 2. Quite often
 - 3. Frequently

If you have experienced sudden episodes of crying, please answer the following (questions 11–18).

11. Have these episodes occurred without any cause in your surroundings?
- ___ Rate the frequency with which the episodes have occurred without external stimuli in the past two weeks.
- 0. Rarely or not at all
 - 1. Occasionally
 - 2. Quite often
 - 3. Frequently
12. Have these episodes lasted for a long period of time?
- ___ Rate the average duration of the episodes during the past two weeks.
- 0. Very brief
 - 1. Short (a few seconds)
 - 2. Moderate (less than 30 seconds)
 - 3. Prolonged (more than 30 seconds)
13. Have these episodes been uncontrollable by you?
- ___ Rate the ability to control the episodes during the past two weeks.
- 0. Rarely or not at all
 - 1. Occasionally
 - 2. Quite often
 - 3. Frequently

14. Have these episodes occurred as a result of feelings of sadness?
- ___ Rate the frequency with which the episodes have occurred as a result of sadness in the past two weeks. The sadness must precede or accompany the crying and not be a reaction to it.
- 0. Rarely or not at all
 - 1. Occasionally
 - 2. Quite often
 - 3. Frequently
15. Have these episodes occurred in excess of feelings of sadness?
- ___ Rate the frequency with which the episodes have been disproportionate to the emotional state in the past two weeks
- 0. Rarely or not at all
 - 1. Occasionally
 - 2. Quite often
 - 3. Frequently
16. Have these episodes of crying occurred with feelings of happiness?
- ___ Rate the frequency of association between the episode and the paradoxical emotion in the past two weeks. The happiness must precede or accompany the crying.
- 0. Rarely or not at all
 - 1. Occasionally
 - 2. Quite often
 - 3. Frequently
17. Have these episodes occurred with any emotions other than sadness or happiness, such as nervousness, anger, fear, etc.?
- ___ Rate the frequency of association between the episodes and emotions in the past two weeks. The emotions must precede or accompany the episode and not be a reaction to it.
- 0. Rarely or not at all
 - 1. Occasionally
 - 2. Quite often
 - 3. Frequently
18. Have these episodes caused you any distress or social embarrassment?
- ___ Rate the degree of distress or embarrassment caused by the episodes in the past two weeks.
- 0. Rarely or not at all
 - 1. Occasionally
 - 2. Quite often
 - 3. Frequently

Depressive Symptoms and the Self-Reported Use of Alcohol, Caffeine, and Carbohydrates in Normal Volunteers and Four Groups of Psychiatric Outpatients

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Objective: The authors examined the relationship between depressive symptoms and the self-reported use of alcohol, carbohydrates, and caffeine in normal volunteers and four groups of psychiatric outpatients. **Method:** Outpatients and normal volunteers were given a questionnaire asking about their use of each of the three substances in response to each of the 14 depressive symptoms on the Hamilton Rating Scale for Depression. They also rated how much each substance improved each symptom. Twenty-six normal volunteers, 35 patients with major depression, 117 patients with seasonal affective disorder, 16 patients with alcohol dependence, and 24 patients with comorbid primary depression and secondary alcohol dependence completed the questionnaire. Test-retest reliability was established. Analysis of variance and stepwise multivariate discriminant function analyses were used to determine if diagnostic groups differed in the reported use and effect of each of the three substances. **Results:** The responses concerning use and effect of alcohol of patients with alcohol dependence with or without depression were indistinguishable from each other. The responses of the patient groups regarding caffeine and carbohydrate use did not differ from each other, but all differed significantly from the responses of normal volunteers. Discriminant function analysis distinguished alcoholics from nonalcoholics in the relationship between drinking and the symptoms of anger and anhedonia. **Conclusions:** The relationship between symptoms and substance use varied depending on the substance. Alcoholics without depression were as likely to report drinking in response to depressive symptoms as were those who had had depression. Patients of all diagnostic groups were more likely than normal volunteers to report using caffeine and carbohydrates in response to depressive symptoms.

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A complex relationship exists between psychiatric illness and the use of nonprescribed psychoactive substances. When a suffering person ingests a psychoactive substance, there are at least three ways to understand the coexistence of the person's symptoms and substance use. First, it is possible that the symptom and substance use are independent, each with its own probability. For example, since approximately 10% of the population has major affective disorder and 10% of the population is alcoholic, chance alone would dictate that 1% of the population would be depressed and drink to excess (1). Second, habitual use of the substance may have caused the patient's symptoms. Alcoholic binges frequently cause secondary de-

pression (2), and the symptoms of caffeinism may mimic those of panic disorder (3).

The "self-medication" hypothesis defines a third possible relationship between psychiatric illness and the use of nonprescribed substances. According to this hypothesis, the patient uses the substance in an attempt to obtain relief from the symptoms. For example, patients with seasonal affective disorder, who experience psychomotor retardation when depressed, crave carbohydrates and feel energized after they ingest them (4-7). Both children and adults who use high levels of caffeine appear to be lethargic at baseline and may therefore be using caffeine's stimulant properties to "treat" their hypoarousal (8, 9).

Despite the plausibility of the self-medication hypothesis, it is difficult to demonstrate in a systematic way. For example, although clinicians frequently claim that patients treat their depression with alcohol, researchers have not been able to demonstrate this phenomenon (10-16). This disparity between clinical anec-

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dote and systematic research may result from the fact that heterogeneous groups of depressive subjects have been used in most studies. Given that both depression and alcoholism are probably heterogeneous illnesses (17, 18) and that there is wide interindividual variation in subjects' responses to alcohol (19–21), such an approach may have missed a subgroup of patients in whom depression does predispose to alcoholism.

Studies on self-medication have generally assumed a simple causal relationship between psychopathology and substance abuse. For example, many studies have implicitly assumed that the extent of a patient's depression is the sole determinant of that patient's drinking behavior (10, 16). It may be more useful to view depressive symptoms as one of a number of stimuli that increase craving. Sherman et al. (22) suggested that drug craving may be precipitated by a negative mood state but that other cognitive processes (e.g., attributing dysphoria to drug withdrawal) must be present for the craving to culminate in drug consumption.

Self-medication alone, therefore, is probably not a sufficient explanation for a psychiatric patient's addiction. Nonetheless, the self-medication hypothesis may have important implications for patient care and for our understanding of the pathophysiology of mood disorders. For example, self-medication may help to explain a patient's "drug of choice" for abuse. Self-medication may serve as a naturalistic probe into the biochemical abnormalities of depression, in that a patient's choice of psychoactive substance may provide a clue to the neurochemistry underlying his or her symptoms. In addition, understanding self-medication may help to clarify the circumstances under which relapse is likely to occur. If the symptoms leading to self-medication can be defined, clinicians can target treatment toward those symptoms and decrease the likelihood of relapse. Clinicians can also recognize times when the patient is especially likely to relapse and intensify treatment in order to forestall clinical deterioration.

We decided to study the question of self-medication in a way that differs substantially from previous approaches. First, we studied several diagnostic groups for comparative purposes. We studied alcohol-dependent patients with no history of depression, patients with primary depression and secondary alcohol dependence, patients with depression and no history of substance abuse, patients with seasonal affective disorder (a homogeneous subgroup of depression) (4), and normal volunteers. Second, we reasoned that "depression" is a physician's construct; particular symptoms, rather than depression per se, might motivate a patient to treat these symptoms ("self-medicate") with a psychoactive substance. Therefore, we studied patients' reported substance use and the effect of these substances as they related to specific depressive symptoms rather than to global measures of depression.

Third, we asked patients about their use of three psychoactive substances to determine if they used particular substances to treat specific symptoms. Given the literature concerning the response of patients with sea-

sonal affective disorder to carbohydrates, we included carbohydrates as a psychoactive substance in our questionnaire, along with caffeine and alcohol.

Finally, we asked subjects about the effect that they believed each substance had on each depressive symptom. We could then determine the extent to which each subgroup reported experiencing the desired effect from each substance.

METHOD

In addition to normal volunteers, outpatients in four diagnostic categories were recruited for participation in the study: patients with nonseasonal major depression, patients with seasonal affective disorder, patients with alcohol dependence without a history of depression, and patients with comorbid primary major depression and secondary alcohol dependence. Patients with seasonal affective disorder and patients with major depression were participating in ongoing research at the National Institute of Mental Health and had been interviewed with the Structured Clinical Interview for DSM-III-R (SCID) (23). Patients with psychoactive substance use disorders were excluded from these groups. Normal volunteers, patients with alcohol dependence, and patients with comorbid primary depression and secondary alcohol dependence were recruited through local media and support groups. Diagnoses were established either by personal interview with the SCID or by telephone interview with a modified telephone version of the SCID, including the psychoactive substance use and affective disorder sections. In the case of the patients with comorbid depression and alcohol dependence, the interviewer ascertained that the onset of major depression preceded the alcohol dependence and that the patient had had episodes of depression even when sober. Patients in the alcohol dependence and comorbid depression and alcohol dependence groups had been sober for at least 3 months at the time of recruitment.

Subjects were asked to complete a questionnaire concerning their use of carbohydrates, alcohol, and caffeine in response to each of 14 depressive symptoms (sluggishness, insomnia, guilt, poor appetite, worry, anxiety, anhedonia, low self-esteem, decreased concentration, sadness, pessimism, social discomfort, anger, and decreased libido). The symptoms were chosen to correspond with those of the Hamilton Rating Scale for Depression (24), and the wording used on the individual items was similar to that of the Hamilton scale. Patients with major depression and patients with seasonal affective disorder were asked to respond to the questionnaire in terms of their behavior when they experienced acute depression. In addition, patients were asked to rate the degree to which each substance improved each of the depressive symptoms. A 7-point Likert scale was used (1=not at all and 7=very much). Thus, for each substance, patients rated each symptom twice on the 7-point scale: once to indicate the likelihood of using the substance to treat the symptom, and

TABLE 1. Frequency Distribution of Kappa Values for 20 Normal Volunteers and 87 Patients With Seasonal Affective Disorder Who Completed Substance Use Questionnaire Twice

Kappa	Strength of Agreement	Number of Items		
		Normal Volunteers (N=84) ^a	Patients (N=84) ^a	Total (N=168) ^a
<0.00	Poor	3	0	3
0.00–0.20	Slight	7	0	7
0.21–0.40	Fair	11	0	11
0.41–0.60	Moderate	28	1	29
0.61–0.80	Substantial	30	40	70
0.81–1.00	Almost perfect	5	43	48

^aNumber of use and effect items for alcohol, caffeine, and carbohydrates.

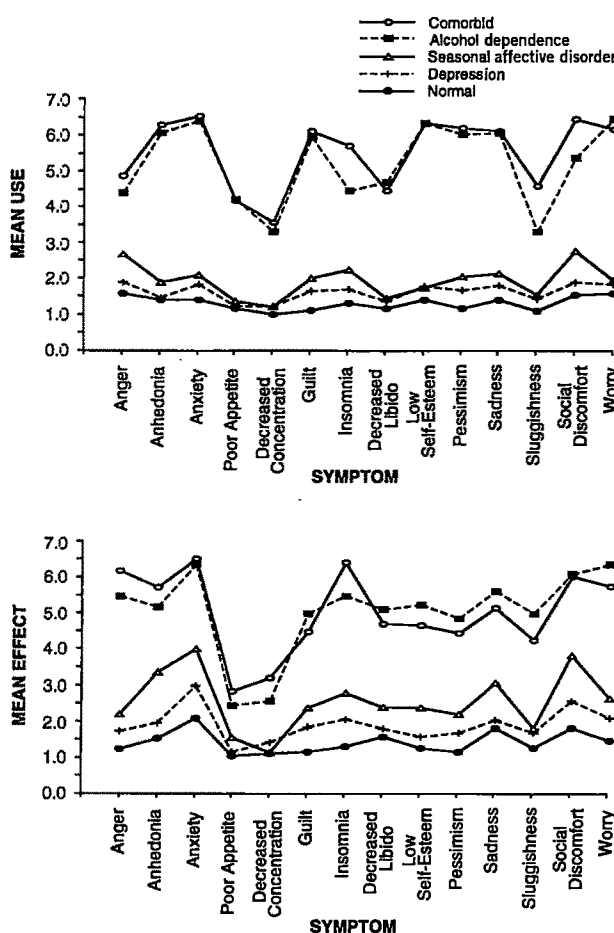
once to rate the extent to which the substance improved the symptom. Patients were also asked to indicate the amount of caffeinated beverages and alcohol that they consumed during the summer months and during the winter months.

The 218 subjects who completed the questionnaire included 26 normal volunteers, 35 patients with major depression, 117 patients with seasonal affective disorder, 16 patients with alcohol dependence, and 24 patients with comorbid primary depression and secondary alcohol dependence. Fifty-four (25%) of the subjects were men and 164 (75%) were women.

To establish test-retest reliability, a subset of the normal volunteers (N=20) and patients with seasonal affective disorder (N=87) completed the questionnaire twice, with a 2-week interval, with no treatment intervention between the two test administrations. The weighted version of the coefficient of agreement, kappa, was computed by using a FORTRAN program (25–27). The reported strength of agreement for each kappa was based on guidelines suggested by Landis and Koch (28). Table 1 shows the frequency distribution of the kappa values for all use and effect items across all three substances for these subjects. Overall, the responses of the normal volunteers showed moderate agreement for the alcohol (kappa=0.53) and carbohydrate (kappa=0.53) items and substantial agreement (kappa=0.70) for the caffeine items. Kappa values for the responses of the patients with seasonal affective disorder were in the almost perfect range for all three substances (kappa=0.85 for alcohol, 0.84 for caffeine, and 0.81 for carbohydrates).

So that the results would not be disproportionately affected by the large number of patients with seasonal affective disorder, the remaining analyses used a random sample of 25 of the 117 patients with seasonal affective disorder. The gender distribution of these 25 patients was representative of the entire group of patients with seasonal affective disorder.

The data were analyzed as six separate data sets, comprising use and effect data for each of the three substances. Use data reflect the self-reported likelihood that subjects would use a substance to treat each of the

FIGURE 1. Use and Effect of Alcohol in Response to Depressive Symptoms Reported by Normal Volunteers and Four Groups of Psychiatric Outpatients^a

^aAnalyses were conducted with 26 normal volunteers, 35 patients with major depression, 25 patients with seasonal affective disorder, 16 patients with alcohol dependence, and 24 patients with comorbid primary depression and secondary alcohol dependence. The numbers on the vertical axis correspond to the 7-point Likert scale used in the questionnaire: 1=not at all and 7=very much.

depressive symptoms. Effect data reflect the self-reported likelihood that subjects would experience a beneficial effect on the symptom from the substance. To determine if the use and effect data differed between diagnostic groups, each data set was analyzed by using repeated-measures analyses of variance (ANOVAs) with Greenhouse-Geisser corrections and post hoc *t* tests with Bonferroni corrections. These analyses were performed by using BMDP and Systat software. Significance was set at *p*<0.05.

Where the ANOVAs revealed significant differences between diagnostic groups, we attempted to further differentiate among the groups by identifying those specific symptoms believed by patients in each group to motivate substance use or to cause them to respond to each psychoactive substance. To accomplish this, we performed stepwise multivariate discriminant function

TABLE 2. Results of Analyses of Variance of Use and Effect of Alcohol, Caffeine, and Carbohydrate in Response to Depressive Symptoms Reported by Normal Volunteers and Four Groups of Psychiatric Outpatients^a

Variable	Between-Group Difference			Within-Group Difference			Group by Symptom Interaction		
	F	df	p	F	df ^b	p	F	df ^b	p
Alcohol use									
All subjects	96.76	4, 117	<0.0001	23.99	1, 117	<0.0001	4.47	1, 30	<0.0001
Alcohol dependence versus comorbid	0.77	1, 38	n.s.	15.20	1, 38	<0.01	0.95	1, 38	n.s.
Seasonal affective disorder versus major depression versus normal	2.81	2, 79	n.s.	8.58	1, 79	<0.01	1.24	1, 40	n.s.
Combined alcohol dependence and comorbid versus combined seasonal affective disorder, major depression, and normal	372.35	1, 120	<0.01	28.77	1, 120	<0.01	14.68	1, 120	<0.01
Alcohol effect									
All subjects	49.48	4, 118	<0.0001	40.63	1, 118	<0.0001	4.14	1, 30	<0.0001
Alcohol dependence versus comorbid	0.07	1, 37	n.s.	18.57	1, 37	<0.01	1.21	1, 31	n.s.
Major depression versus normal	3.69	1, 57	n.s.	10.76	1, 57	<0.01	1.09	1, 57	n.s.
Caffeine use									
All subjects	6.15	4, 115	<0.0005	49.04	1, 115	<0.0001	1.36	1, 26	n.s.
Seasonal affective disorder versus alcohol dependence versus major depression	0.65	2, 67	n.s.	28.87	1, 67	<0.01	0.80	1, 34	n.s.
Combined seasonal affective disorder, alcohol dependence, and major depression versus comorbid	5.81	1, 92	n.s.	32.92	1, 93	<0.01	0.59	1, 92	n.s.
Combined seasonal affective disorder, alcohol dependence, major depression, and comorbid versus normal	15.23	1, 118	<0.01	24.41	1, 118	<0.01	2.93	1, 118	n.s.
Caffeine effect									
All subjects	7.13	4, 117	<0.0001	53.63	1, 117	<0.0001	2.04	1, 29	<0.001
Seasonal affective disorder versus alcohol dependence versus major depression	1.48	2, 69	n.s.	31.08	1, 69	<0.01	1.06	1, 35	n.s.
Carbohydrate use									
All subjects	9.01	4, 116	<0.001	25.58	1, 116	<0.0001	1.62	1, 29	<0.05
Seasonal affective disorder versus alcohol dependence versus major depression versus comorbid	1.11	3, 91	n.s.	22.17	1, 91	<0.01	1.06	1, 30	n.s.
Combined seasonal affective disorder, alcohol dependence, major depression, and comorbid versus normal	31.85	1, 119	<0.01	11.91	1, 119	<0.01	2.77	1, 119	n.s.
Carbohydrate effect									
All subjects	5.41	4, 116	<0.001	21.52	1, 116	<0.0001	1.74	1, 29	<0.005

^aAnalyses were conducted with 26 normal volunteers, 35 patients with major depression, 25 patients with seasonal affective disorder, 16 patients with alcohol dependence, and 24 patients with comorbid primary depression and secondary alcohol dependence.

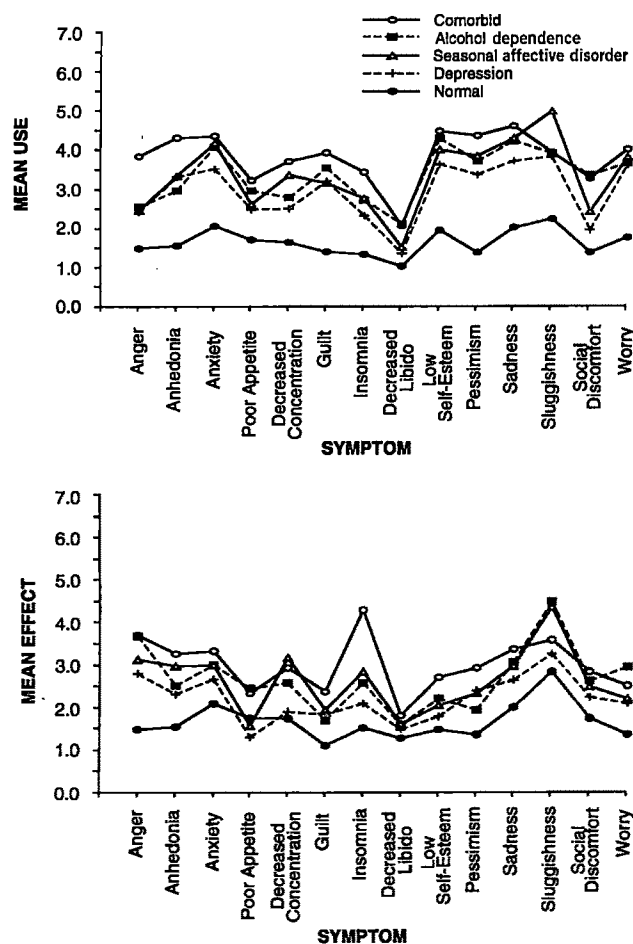
^bGreenhouse-Geisser-corrected degrees of freedom.

analyses excluding the normal volunteers. The BMDP program was run with an "F-to-enter" setting of 4.00, and a jackknifed-validation procedure was used.

RESULTS

Figure 1 shows the reported use of alcohol in response to each of the depressive symptoms by patients in each of the diagnostic categories. A two-way ANOVA showed significant effects between diagnostic groups, within groups, and for a Group by Symptom interaction (F, df, and p values for all reported ANOVAs are given in table 2).

To further identify the differences between diagnostic groups, post hoc ANOVAs were performed. Groups that did not show a significant between-group effect were then combined for further comparisons. There was no significant difference in the reported use of alcohol in response to depressive symptoms in the comparison of the patients with alcohol dependence versus those with comorbid depression and alcohol dependence or in the comparison of the patients with seasonal affective disorder versus the normal volunteers versus the patients with major depression (table 2). An ANOVA with the patients with alcohol dependence and the patients with comorbid depression and alcohol dependence combined in one group and the patients

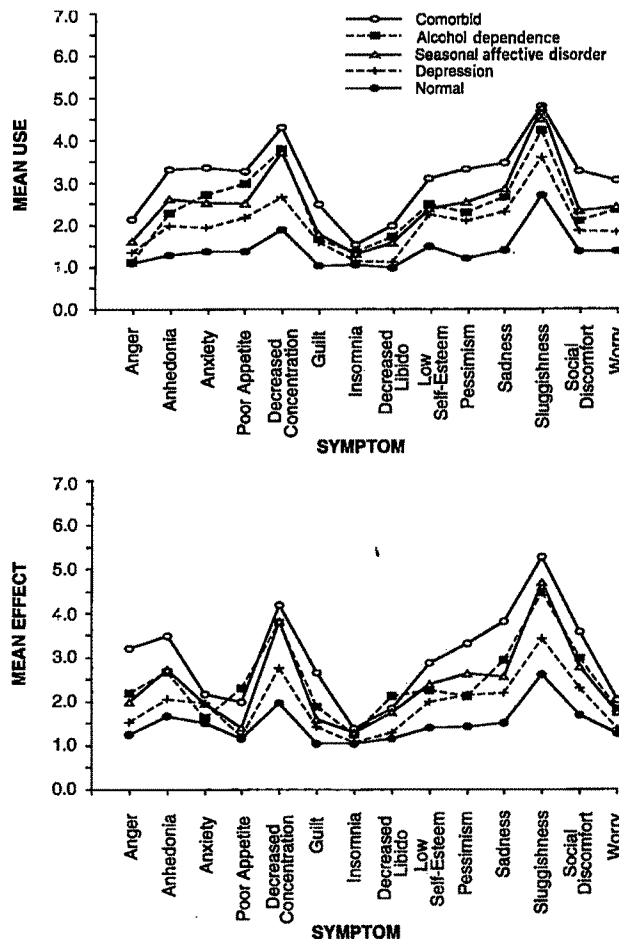
FIGURE 2. Use and Effect of Caffeine in Response to Depressive Symptoms Reported by Normal Volunteers and Four Groups of Psychiatric Outpatients^a

^aAnalyses were conducted with 26 normal volunteers, 35 patients with major depression, 25 patients with seasonal affective disorder, 16 patients with alcohol dependence, and 24 patients with comorbid primary depression and secondary alcohol dependence. The numbers on the vertical axis correspond to the 7-point Likert scale used in the questionnaire: 1=not at all and 7=very much.

with seasonal affective disorder, the normal volunteers, and the patients with major depression in a second group, however, showed a significant between-group difference. Post hoc *t* tests were significant at the $p < 0.001$ level for each of the 14 symptoms.

The responses for the effect of alcohol on depressive symptoms also showed significant main effects between and within groups as well as a Group by Symptom interaction (table 2 and figure 1). Post hoc ANOVAs showed no significant difference between the patients with alcohol dependence and the patients with comorbid depression and alcohol dependence or between the normal volunteers and the patients with major depression.

The ANOVA for the use of caffeine showed significant between-group and within-group effects, but the Group by Symptom interaction was not significant (ta-

FIGURE 3. Use of Carbohydrates in Response to Depressive Symptoms Reported by Normal Volunteers and Four Groups of Psychiatric Outpatients^a

^aAnalyses were conducted with 26 normal volunteers, 35 patients with major depression, 25 patients with seasonal affective disorder, 16 patients with alcohol dependence, and 24 patients with comorbid primary depression and secondary alcohol dependence. The numbers on the vertical axis correspond to the 7-point Likert scale used in the questionnaire: 1=not at all and 7=very much.

ble 2 and figure 2). The between-group effect for the four patient groups was not significant. When the four patient groups were combined into one group and compared with the normal volunteers, there was a significant between-group effect.

The overall ANOVA for the effect of caffeine showed significant between-group and within-group effects as well as a significant Group by Symptom interaction. There was no significant difference between the patients with seasonal affective disorder, alcohol dependence, and major depression.

The responses for the use of carbohydrates (table 2 and figure 3) showed a similar pattern to those concerning the use of caffeine. The overall ANOVA showed significant between- and within-group effects and a significant Group by Symptom interaction. The four patient groups did not differ significantly from each other,

but when they were combined into one group and compared with the normal volunteers there was a significant between-group effect.

The overall ANOVA for the effect of carbohydrates showed significant between- and within-group effects and a Group by Symptom interaction. Post hoc analyses were not performed because subgroups were not evident.

Stepwise discriminant function analysis was then used to identify specific symptoms that could distinguish among the patient groups. For these analyses, the group comparisons already described were used to justify combining patient groups that did not differ from each other. Thus, for the use and effect of alcohol, the patients with seasonal affective disorder and the patients with major depression were combined into one group, and the patients with alcohol dependence and those with comorbid depression and alcohol dependence were combined into a second group. For alcohol use, responses to the anhedonia item correctly classified 91.1% of the patients in the seasonal affective disorder-major depression group and 90.0% of the patients in the alcohol dependence-comorbid depression and alcohol dependence group ($F=222.81$, $df=1, 94$, $p<0.001$). However, six other symptoms (anxiety, low self-esteem, worry, pessimism, sadness, and guilt) also had high F values, ranging from 161.67 for guilt to 218.72 for anxiety. The jackknifed validation procedure identified pessimism as the second discriminating symptom, but this added only 1.9% more correctly identified subjects to the seasonal affective disorder-major depression group and no more subjects to the alcohol dependence-comorbid depression and alcohol dependence group.

For alcohol effect, responses to the anger item correctly classified 87.9% of the patients in the seasonal affective disorder-major depression group and 87.2% of the patients in the alcohol dependence-comorbid depression and alcohol dependence group. Social discomfort was the second symptom identified, but again it added little to the number of patients correctly classified.

Since there were no significant differences between patient groups in the reported use or effect of caffeine and carbohydrates, the results of the discriminant function analyses for these substances will not be reported.

DISCUSSION

The idea that people take drugs to relieve unpleasant feelings was first explicitly stated by Conger (29, 30), who theorized that alcoholics drink to relieve anxiety. More recently, the concept has been studied in terms of the negative reinforcement properties of addictive substances—in other words, the degree to which addicts ingest a substance because it relieves negative affect (22, 31–33). However, these studies involved addicts without other psychopathology. In contrast, our study was designed to document systematically the relationship between symptoms and substance ingestion in several psychiatric groups.

When subjects were asked about their alcohol use in response to depressive symptoms, the responses of the patients with alcohol dependence and those with comorbid depression and alcohol dependence were indistinguishable. Thus, patients with alcohol dependence who did not suffer from clinical depression reported using alcohol to “treat” depressive symptoms to the same degree as did patients with diagnosable depression. Similarly, nonalcoholic patients (the patients with seasonal affective disorder, the patients with major depression, and the normal volunteers) did not differ from each other in their responses. A history of more severe depressive symptoms was not necessarily associated with patients’ reporting that they were more likely to drink in response to their symptoms.

For each depressive symptom, alcohol-dependent patients were more likely than non-alcohol-dependent patients to drink in response to that symptom. However, there was also a within-group effect, indicating that alcoholics were more likely to use alcohol in response to some symptoms than others. On the discriminant function analysis, drinking to relieve anhedonia was best able to distinguish subjects with both primary and secondary alcohol dependence from subjects without alcohol dependence. Thus, alcoholics may perceive themselves as being unable to experience pleasure in the sober state.

The alcohol-dependent subjects were more likely than the other subjects to report that alcohol relieved each of the depressive symptoms. Other studies have demonstrated that individuals differ in their response to alcohol and that these differences may predict alcohol use (20, 21). Using discriminant function analysis, we could distinguish the alcohol-dependent subjects in our study group from the rest of the subjects by the degree to which they reported that drinking relieved anger and social anxiety. Other studies have indicated a relationship between alcohol dependence and anger (15, 34, 35) and between alcohol dependence and social phobia (36). In our study, the alcohol-dependent subjects may have reported different responses to alcohol because of neurophysiological factors that predated the development of alcohol dependence or because of the sequelae of drinking. That is, alcohol dependence itself may alter a patient’s response to alcohol, either through biological changes, conditioning, or other mechanisms (19, 32).

When asked about their use of caffeine in response to depressive symptoms, the patients with a psychiatric diagnosis (alcohol dependence, comorbid depression and alcohol dependence, major depression, or seasonal affective disorder) did not differ from each other in their responses but did differ significantly from normal volunteers. This result has several possible explanations. Because they experience distressing symptoms, patients may be more aware of their internal state. Therefore, they may be more likely to use a substance in response to internal cues. In addition, since most of these patients have received psychiatric treatment, their responses may reflect a learned tendency to relate their behavior to their affective state.

The reported use of carbohydrates was similar to the

reported use of caffeine in that the patient groups did not differ from each other but did differ significantly from the normal volunteers. Patients in distress may have a somewhat nonspecific response to their symptoms, turning to carbohydrates, caffeine, and perhaps other substances as they seek relief. However, we found that their response has some specificity in that only the subjects with primary and secondary alcohol dependence tended to report using alcohol in response to their symptoms.

The results for carbohydrates indicate that the use of carbohydrates in response to depressive symptoms may be characteristic of a variety of patient groups. Carbohydrate craving has been thought to be characteristic of patients with atypical depression (37), seasonal affective disorder (4, 5), and premenstrual syndrome (38, 39). Frank et al. (40) suggested that increased appetite is a common feature of depression in women, cutting across a variety of subtypes. Our results suggest that the use of carbohydrates in response to depressive symptoms may be quite general, including a number of depressive subtypes as well as alcohol dependence.

The validity of our results is limited by the subjective and retrospective nature of our methods. Thus, although patients reported that they are likely to use a given substance when they experience particular symptoms, we do not have prospective, external validation that they actually do so. However, the marked difference in the reported use of alcohol in response to symptoms between the alcohol-dependent patients and the other patients provides face validity to the measure.

Further exploration of the self-medication hypothesis will require prospective studies that track substance use in conjunction with externally validated symptom measures. However, even these studies will be unable to distinguish whether patients actually ingest substances in response to symptoms or simply attribute their substance use to self-medication when the symptoms and substance use are not causally related. Only controlled intervention studies, where investigators manipulate target symptoms while monitoring how substance use varies with patients' symptoms, can resolve this question definitively.

What are the implications of our findings for the self-medication hypothesis? Clearly, the reported relationship between symptoms and substance use varies depending on the substance in question. Our results for alcohol differed markedly from those for caffeine and carbohydrates. Alcohol-dependent subjects with no history of depression were as likely to report drinking in response to depressive symptoms as were alcohol-dependent subjects who had episodes of primary depression. In this addicted population, drinking may occur regardless of the presence or severity of symptoms and yet be attributed to self-medication, or subclinical symptoms may be sufficient to precipitate use.

In the case of carbohydrates and caffeine, patients of all diagnostic groups reported using these less addictive substances in response to depressive symptoms. As noted, this may indicate a nonspecific form of self-

medication or may reflect a difference in attribution patterns between psychiatric patients and normal volunteers. Studies of the sort already suggested would help to resolve these questions and to increase our understanding of the factors motivating depressed patients to ingest psychoactive substances.

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Body Dysmorphic Disorder: 30 Cases of Imagined Ugliness

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Objective: Body dysmorphic disorder, preoccupation with an imagined defect in appearance, is included in DSM-III-R but has received little empirical study. The authors investigated the demographics, phenomenology, course, associated psychopathology, family history, and response to treatment in a series of 30 patients with the disorder. **Method:** The patients (including 12 whose preoccupation was of probable delusional intensity) were assessed with a semistructured interview and the Structured Clinical Interview for DSM-III-R, and their family histories were obtained. **Results:** The 17 men and 13 women reported a lifetime average of four bodily preoccupations, most commonly "defects" of the hair, nose, and skin. The average age at onset of body dysmorphic disorder was 15 years, and the average duration was 18 years. Seventy-three percent of the patients reported associated ideas or delusions of reference; 73%, excessive mirror checking; and 63%, attempts to camouflage their "deformities." As a result of their symptoms, 97% avoided usual social and occupational activities, 30% had been housebound, and 17% had made suicide attempts. Ninety-three percent of the patients had an associated lifetime diagnosis of a major mood disorder; 33%, a psychotic disorder; and 73%, an anxiety disorder. The patients generally responded poorly to surgical, dermatologic, and dental treatments and to adequate trials of most psychotropic medications, with the exception of fluoxetine and clomipramine (to which more than half had a complete or partial response). **Conclusion:** This often secret, chronic disorder can cause considerable distress and impairment, may be related to obsessive-compulsive disorder or mood disorder, and may respond to serotonin reuptake-blocking antidepressants.

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Body dysmorphic disorder, preoccupation with an imagined defect in physical appearance (for example, a large nose, "elfish" ears, or small genitals), has long been described in the European, Russian, and Japanese literature under a variety of names, most commonly dysmorphophobia (1-7), but it has received very little empirical attention. Dysmorphophobia first entered the official psychiatric nomenclature as a separate disorder in DSM-III-R, where its name was changed to body dysmorphic disorder. Body dysmorphic disorder is considered to be non-delusional and is classified as a somatoform disorder whereas delusional body dysmorphic disorder is considered to be a type of delusional disorder, somatic type (which is similar to mono-

symptomatic hypochondriacal psychosis [8]), and is classified as a psychotic disorder.

Despite its century-long description in the literature, surprisingly little is known about body dysmorphic disorder. In a recent review (9), we found no systematic studies of the phenomenology, associated psychopathology, family history, or treatment history of individuals rigorously diagnosed as having body dysmorphic disorder. We therefore assessed these features in a series of 30 consecutive patients with body dysmorphic disorder.

METHOD

Originally, 32 consecutive patients with apparent body dysmorphic disorder were obtained for the study from our own practices (N=11) and from referrals by clinicians at McLean Hospital (N=21). All patients met DSM-III-R criterion A for body dysmorphic disorder ("preoccupation with some imagined defect in appearance in a normal-appearing person; if a slight physical anomaly is present, the person's concern is grossly excessive") and criterion C ("occurrence not exclusively

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during the course of anorexia nervosa or transsexualism"). The 12 patients who failed to meet criterion B (i.e., they had preoccupations that were of delusional intensity) were also included. Two of the 32 patients were excluded from the final study group; one failed to complete the interview, and one had relatively normal (i.e., not significantly distressing or impairing) concerns about his appearance.

We administered a semistructured interview to elicit demographic data and information on the phenomenology, age at onset, course, associated features, treatment history, and response to treatment of the disorder, followed by the Structured Clinical Interview for DSM-III-R (SCID) (10). Family histories were obtained from the patients by an investigator who was blind to their diagnosis; blindness was maintained by including this study in a larger family history study of patients with a diverse array of psychiatric disorders. Morbid risk was calculated by the Weinberg shorter method (11), with ages 16–65 years as the period of risk for the development of a major mood disorder.

This historical information was supplemented by a review of the inpatient records of the 17 patients who had been hospitalized at McLean Hospital and the outpatient records of the additional six patients for whom such records were available. For patients treated by us (N=14), response to treatment (percent improvement) was based on our assessment and that of the patients; for the other subjects, it was based on retrospective reports of the patients and, when available, clinicians' reports and record reviews.

RESULTS

The 30 patients had a mean age of 33.2 years (SD=11.3, range=17–80). Fifty-seven percent (N=17) were male. Eighty-three percent (N=25) had never been married, 7% (N=2) were divorced, and 10% (N=3) were married. Most patients (57%, N=17) were unemployed, 17% (N=5) worked part-time, 7% (N=2) were students, and 20% (N=6) had full-time jobs. Fewer than half were living independently; 37% (N=11) were living with their parents, and 17% (N=5) were living in a halfway house or other supervised setting.

Symptoms of body dysmorphic disorder focused on a wide array of body parts but overwhelmingly involved the face or head. In fact, all but two patients (93%) had at least one such concern (table 1). With the exception of two patients, who had minimal anomalies about which they were excessively preoccupied, all body parts of concern appeared normal to the investigators. Many concerns were quite specific—for example, a bumpy nose, a crooked lip, an egg-shaped head, or thinning hair—but others were notably vague, such as inadequately firm eyes, atrophied facial muscles, or an ugly face. Nearly all patients (87%, N=26) had histories of multiple symptoms (mean=4.3 lifetime symptoms, SD=2.6, range=1–13), most of which were present concurrently at the time of evaluation. However, this aver-

TABLE 1. Location of Imagined Defects in 30 Patients With Body Dysmorphic Disorder^a

Location	N	%
Hair ^b	19	63
Nose	15	50
Skin ^c	15	50
Eyes	8	27
Head/face ^d	6	20
Overall body build/bone structure	6	20
Lips	5	17
Chin	5	17
Stomach/waist	5	17
Teeth	4	13
Legs/knees	4	13
Breasts/pectoral muscles	3	10
Ugly face (general)	3	10
Ears	2	7
Cheeks	2	7
Buttocks	2	7
Penis	2	7
Arms/wrists	2	7
Neck	1	3
Forehead	1	3
Facial muscles	1	3
Shoulders	1	3
Hips	1	3

^aTotal is greater than 100% because most patients had "defects" in more than one location.

^bInvolved head hair in 15 cases, beard growth in two cases, and other body hair in three cases.

^cInvolved acne in seven cases, facial lines in three cases, and other skin concerns in seven cases.

^dInvolved concerns with shape in five cases and size in one case.

age may be an underestimate, as most patients were extremely ashamed of and embarrassed by their concerns and reluctant to discuss them with the investigators.

To convey the intensity of their preoccupation, the patients used such terms as "obsessed," "fixated," "devastated," and "tormented." Most spent at least several hours a day thinking about their "defects," and some said that this concern dominated their lives, claiming that they thought about it "all day, every day."

Indeed, many patients' concerns had persisted for much of their lives. The average age at onset of body dysmorphic disorder was 14.8 years (SD=6.4, range=6–33), and the course of illness tended to be chronic, with an average duration of 18.3 years (SD=13.1). Some patients had concerns that persisted unchanged, whereas others added new "defects" to preexisting ones. A third common pattern consisted of complex additions and remissions of various symptoms, often with replacement of one by another. With all three patterns, the intensity of symptoms often waxed and waned over time, but all symptoms rarely remitted fully.

Associated symptoms and behaviors included ideas or delusions of reference related to the "defect" in 73% (N=22) of the cases and subtle tactile sensations, such as facial tightness, in 37% (N=11). Seventy-three percent (N=22) of the patients excessively checked their appearance in mirrors and other reflecting surfaces—often car bumpers and car and store windows—for up to 4 hours a day. Eight of these patients also peri-

TABLE 2. Current and Lifetime DSM-III-R Diagnoses of 30 Patients With Body Dysmorphic Disorder

DSM-III-R Diagnosis	Current		Lifetime	
	N	%	N	%
Mood disorders ^a	23	77	28	93
Major depression	15	50	18	60
Bipolar disorder	8	27	10	33
Dysthymia ^b	2	7	—	—
Psychotic disorders	9	30	10	33
Delusional disorder, somatic type	2	7	3	10
Schizophrenia	0	0	0	0
Schizoaffective disorder	1	3	1	3
Psychotic disorder not otherwise specified	6	20	6	20
Anxiety disorders ^a	21	70	22	73
Panic disorder	3	10	5	17
Agoraphobia without panic	1	3	2	7
Social phobia	13	43	15	50
Simple phobia	1	3	1	3
Obsessive-compulsive disorder	9	30	11	37
Psychoactive substance use disorders (abuse or dependence) ^a	6	20	14	47
Alcohol	3	10	12	40
Other	3	10	10	33
Somatoform disorders ^b	1	3	—	—
Eating disorders ^a	1	3	3	10
Anorexia nervosa	0	0	2	7
Bulimia nervosa	1	3	2	7

^aThe total is less than the sum of the individual disorders because some subjects had more than one disorder in this category.

^bThe SCID interview assesses only the current (not lifetime) presence of the disorder.

odically avoided mirrors, and another four reported avoidance of mirrors without excessive checking. Several patients repeatedly interrupted the study interview to check their appearance and comb their hair, using the mirror in a nearby bathroom, explaining that they were unable to resist this behavior. Some avoided magazines or television commercials that focused on appearance. Three patients were excessively preoccupied with fears that their "ugly" noses malfunctioned or were extremely fragile and in danger of being damaged, and 33% (N=10) repeatedly questioned others about their appearance. One patient asked, "Dad, do you think I'm losing my hair?" up to 20 times in a half-hour. Most patients (53%, N=19) also attempted to camouflage the imagined defect, growing long hair to cover "asymmetric" beard growth, stuffing their shirts to enhance a "small" penis, or storing candy or wads of paper in the mouth to widen a "thin" face. However, such behavior did little to alleviate their anxiety and distress.

The consequences of body dysmorphic disorder were often profound. Because of embarrassment over their appearance, 97% (N=29) of the patients avoided usual social or occupational activities such as working, shopping, swimming, attending school and gym class, eating, and sex—30% (N=9) to the point of being housebound. Sixty-eight percent (N=15) of the 22 patients who had been psychiatrically hospitalized attributed at least one hospitalization to body dysmorphic disorder; one patient, for example, had been hospitalized because

of his unbearable despair over the failure of Accutane to cure his supposed acne. Twelve patients (40%) had experienced suicidal ideation (23%) or suicide attempts (17%) that they attributed largely or entirely to their distress over their appearance, and one patient had slashed her breasts because she considered them so ugly.

How convinced were patients of their ugliness? That is, did they have insight into the senselessness of their preoccupation, or did it consist of overvalued ideation or even delusional thinking? Although differentiating the subtle gradations of insight proved difficult, it appeared that 53% (N=16) of the patients had overvalued ideas—that is, preoccupations somewhere between delusional and nondelusional thinking. They were not entirely convinced that their defects were real, but they sometimes or often wondered if they were and generally did not consider their concern senseless or "crazy." Forty percent (N=12) of the patients had concerns that appeared delusional, in that they seemed completely convinced that their deformities were real and that their view of them was undistorted. In fact, several tried to convince us of this by showing photographs documenting progressive "ugliness" or by lifting up their sleeves or opening to remove their clothes. Only 7% (N=2) of the patients had good insight into the fact that their defects were imagined rather than real.

Similarly, in nearly all cases, the patients' preoccupations were like DSM-III-R-defined obsessions in that they were recurrent, persistent, and intrusive, but they differed in that they were generally not senseless. (This was also true for the patients with delusional body dysmorphic disorder, except that their concerns were not at all senseless to them.) In nearly all cases, behaviors related to body dysmorphic disorder, such as mirror checking and hair combing, closely conformed to compulsions as defined by DSM-III-R. Several patients with both body dysmorphic disorder and obsessive-compulsive disorder stated that the former was more painful, embarrassing, emotionally distressing, and socially impairing than the latter.

All 30 patients met the DSM-III-R criteria for at least one other psychiatric disorder, with a mood disorder the most common (93%, N=28) (table 2). Depressive episodes tended to be long and persistent; 16 of the 28 patients with a mood disorder reported chronic major depression of an average duration of 14 years. The onset of a mood disorder preceded the onset of body dysmorphic disorder by at least 1 year in three patients (11%), occurred within the same year in nine (32%), and followed it in 16 (57%). Many patients attributed their depression and suicide attempts to their body dysmorphic disorder, and many noted simultaneous worsening and improvement in their symptoms of that disorder and their depressive symptoms.

Seventy-seven percent (N=23) of the patients had lifetime histories of psychotic symptoms, of whom 33% (N=10) had a psychotic disorder and 43% (N=13) a psychotic mood disorder. In nine cases (30% of the series), the psychotic symptoms were entirely attributable to body dysmorphic disorder; six had delusional beliefs

TABLE 3. Treatment History of 30 Patients With Body Dysmorphic Disorder

Treatment	Number of Trials	Response ^a							
		Remission		Partially Improved		Slightly Improved		Worse	
		N	%	N	%	N	%	N	%
Clomipramine ^b	7	2	29	3	43	1	14	0	0
Fluoxetine ^c	19	4	21	6	32	2	11	0	0
Buspirone	3	0	0	2	67	0	0	0	0
Monamine oxidase inhibitors ^d	10	0	0	3	30	0	0	0	0
Tricyclics (excluding clomipramine)	31	0	0	1	3	0	0	1	3
Trazodone	6	0	0	0	0	0	0	0	0
Bupropion	5	0	0	0	0	0	0	0	0
Neuroleptics	44	0	0	0	0	1	2	1	2
Pimozide	4	0	0	0	0	0	0	1	25
Lithium	18	0	0	1	6	0	0	0	0
Anticonvulsants	12	0	0	0	0	0	0	0	0
Benzodiazepines	29	1	3	0	0	0	0	0	0
Stimulants	3	0	0	0	0	0	0	0	0
ECT	4	0	0	0	0	0	0	0	0
Psychotherapy ^e	24	0	0	0	0	2	8	0	0
Behavioral therapy	3	0	0	0	0	0	0	0	0
Surgery/dental work ^f	25	1	4	0	0	1	4	20	80
Dermatologist ^e	6	0	0	0	0	0	0	0	0
Other nonpsychiatric treatment	8	1	12	0	0	1	12	2	25

^aNumbers and percents refer to trials that resulted in each type of response. Trials were deemed to be of adequate dosage and duration; for fluoxetine this consisted of 60 mg/day for 4 weeks, and for clomipramine 200 mg/day for 4 weeks. Remission=90%–100% improvement, partially improved=30%–80% improvement, slightly improved=10%–20% improvement. Trials resulting in no change are not shown in this table.

^bIn three cases percentages reflect the addition of augmenting agents. One slightly improved patient changed to partially improved with the addition of buspirone, one with no improvement changed to partially improved with the addition of fluoxetine (without prior response to fluoxetine alone), and one with no improvement changed to slightly improved with the addition of buspirone (without prior response to buspirone alone).

^cThe average dose producing remission or partial improvement was 62.2 mg/day.

^dOne patient responded only with the addition of amitriptyline.

^eNumbers and percents represent patients rather than trials. Responses in this table refer only to change in body dysmorphic disorder symptoms per se.

^fNonpsychiatric treatment for body dysmorphic disorder was sought by 73% (N=22) of the patients.

about their appearance, and two of these six plus three others had associated delusions of reference. The other 14 patients (47% of the series) had psychosis unrelated to body dysmorphic disorder, although 10 of these 14 also had psychotic symptoms related to the disorder.

Anxiety disorders were also common, particularly social phobia (table 2). The onset of social phobia preceded the onset of body dysmorphic disorder in nearly all patients (N=12 of 15), occurred within the same year in none, and followed it in only three. Obsessive-compulsive disorder, consisting of classic symptoms unrelated to body dysmorphic disorder (such as contamination obsessions and washing compulsions), occurred in 37% (N=11) of the patients. Fourteen patients with a primary anxiety disorder had an additional anxiety disorder apparently secondary to body dysmorphic disorder; these consisted of panic disorder (N=1), agoraphobia (N=5), and social phobia (N=8). Associated non-DSM disorders included olfactory reference syndrome (N=3), erythrophobia (fear of blushing) (N=1), and nondelusional parasitosis (N=1). Surprisingly, comorbidity of body dysmorphic disorder with somatoform and eating disorders was relatively low (table 2).

Given the apparent high comorbidity of body dysmorphic disorder with other psychiatric disorders—as well as

its tendency to be a secret disorder—it is perhaps not surprising that the symptoms of the disorder were mentioned in only five of 17 inpatient chart admission notes or discharge summaries, despite the fact that many patients considered body dysmorphic disorder their most distressing and debilitating problem. And in no chart was body dysmorphic disorder or its DSM-III precursor (atypical somatoform disorder) given as a diagnosis.

Most patients had received many treatments for their symptoms; 28 had received pharmacotherapy (a total of 187 adequate medication trials), 24 had received insight-oriented psychotherapy, three had received behavioral therapy, and four had received ECT (table 3). However, many patients waited years after the onset of body dysmorphic disorder before seeking psychiatric treatment, and even then they often did not divulge the disorder to the persons treating them. In addition, 22 had sought nonpsychiatric treatment, largely from plastic surgeons, dermatologists, and dentists.

Improvement was sometimes difficult to ascribe to a particular treatment modality because patients often received more than one treatment concurrently. With this caveat in mind, we can say that response to most treatments, including most medication trials, was poor. Worth noting is the poor response to neuroleptics; of

TABLE 4. Prevalence of Psychiatric Disorders in the First-Degree Relatives^a of 24 Patients With Body Dysmorphic Disorder

Disorder	Relatives Affected (N=103)	
	N	%
Mood disorders		
Bipolar disorder	1	1
Major depression	18	17
Psychotic disorders	0	0
Anxiety disorders		
Panic disorder and/or agoraphobia	3	3
Obsessive-compulsive disorder	4	4
Eating disorders		
Anorexia nervosa	0	0
Bulimia nervosa	2	2
Alcohol and other psychoactive substance use disorders		
Alcohol	16	16
Other	4	4

^aAge 16 years or older.

the 16 patients receiving neuroleptics, one had a slight response and none had a complete or partial response. The four patients who received an adequate trial of pimozide, reported to be especially effective in the treatment of somatic delusions (12), did not respond. It is notable that 12 of the 16 patients who did not respond to antipsychotics had symptoms of psychotic (delusional) body dysmorphic disorder or associated delusions of reference.

The only medications to which patients consistently responded (58%, N=15) were the serotonin reuptake blockers fluoxetine and clomipramine (table 3). (Of the 26 trials of these two medications, we ourselves conducted 15 and observed an additional three.) Five of the seven patients who received clomipramine and 10 of the 19 who received fluoxetine had a complete or partial reduction in symptoms of body dysmorphic disorder (although these results may underestimate the efficacy of these medications, given the conservative criteria we used for defining an adequate trial) (table 3). The rate of response did not differ significantly between those with comorbid major depression or obsessive-compulsive disorder and those without either of these disorders. Patients with delusional body dysmorphic disorder were just as likely as those with nondelusional body dysmorphic disorder to respond to serotonergic antidepressants, and more than one-half of the responders had previously received neuroleptics without responding.

Responses tended to persist for the duration of treatment, in several cases for 2–3 years. In nine successful trials, medication was discontinued, resulting in sustained remission in one case and relapse in the other eight. In one case, rechallenge with fluoxetine on two occasions resulted in remission of symptoms both times.

Although 73% (N=23) of the patients sought non-psychiatric treatment, most were refused treatment because there was no defect to treat. However, eight patients underwent 25 plastic surgery or dental procedures, most of which led to increased symptoms (table 3).

The prevalence of psychiatric disorders among the patients' first-degree relatives who were 16 years of age or older is presented in table 4. Family data were available for 24 of the 30 patients. The morbid risk for major mood disorders among the first-degree relatives was 28%, and 58% (N=14) of the patients had at least one first-degree relative with a major mood disorder.

DISCUSSION

Many of this study's findings are congruent with the literature on body dysmorphic disorder, particularly our documentation of the intense preoccupation, extreme distress, and profound impairment experienced by these individuals, which can lead to avoidance of many aspects of life, to hospitalization, and to suicide attempts. However, our subjects had a lifetime average of four, and as many as 13, different bodily preoccupations, in contrast to the one or two mentioned in most published case reports. We also found an earlier age at onset—from age 6 through the 20s, with an average of approximately 15 years—in contrast to the ages at onset in case reports—early adolescence through the 20s, with an average of 19 years. In addition, the male-to-female ratio of our patients, 1.3:1, contrasts with the average in case reports in the literature, 1:1.3, but is similar to a large series of Japanese surgical patients with body dysmorphic disorder that was 62% male (13). Many of our patients reported associated tactile sensations, reports of which are largely absent from the literature.

To our knowledge, this is the first study of body dysmorphic disorder to evaluate associated psychopathology with a structured interview and to assess family history systematically. Our results confirm suggestions in the literature (14, 15) that a mood disorder—in particular, major depression—is the disorder most often associated with body dysmorphic disorder and that body dysmorphic disorder often coexists with obsessive-compulsive disorder (16) and social phobia (17, 18). However, in contrast to reports of a high comorbidity of body dysmorphic disorder with schizophrenia (6, 19–21), none of the study patients had schizophrenia, which may reflect the fact that earlier definitions of schizophrenia were overly broad by today's nosologic standards.

Did the study patients actually meet the DSM-III-R criteria for body dysmorphic disorder? All patients met the first criterion, in that they were clearly excessively preoccupied with an imagined defect in appearance, and the concern of the two patients with a true but slight physical anomaly was markedly excessive. However, 12 patients (40%) appeared to have a preoccupation of delusional intensity. Although on the basis of this symptom alone they would qualify for a DSM-III-R diagnosis of delusional disorder, somatic type, most of them received other diagnoses instead because of their chronic and significant mood disorders.

This finding raises the question of whether body dysmorphic disorder and the variant of body dysmorphic

disorder known as delusional disorder, somatic type, are two different disorders or a single disorder. Our results suggest that the two disorders significantly overlap and may even be the same. First, it was difficult to differentiate delusional from nondelusional preoccupations in the majority of the patients, who would best be described as having overvalued ideas—that is, ideas somewhere between delusional and nondelusional thinking. Thus, attempts to clearly differentiate body dysmorphic disorder from delusional disorder are likely to be somewhat arbitrary and unreliable. Second, some patients were at times completely convinced that their deformities were real, but at other times they were not; it is unlikely that they had two different disorders. Third, the 12 patients with delusional concerns did not significantly differ from the patients with nondelusional body dysmorphic disorder in terms of demographic data, phenomenology, associated features, course, associated psychopathology, and response to treatment. Finally, the precursor of body dysmorphic disorder, dysmorphophobia, has historically been classified as both a delusional and a nondelusional disorder (9), and many authors have argued that dysmorphophobia encompasses both psychotic and nonpsychotic conditions (5, 21, 22) or, similarly, that it may be variously expressed as a preoccupation, an obsession, an overvalued idea, or a frank delusion (23). Thus, as Hollander et al. (24) have suggested, it seems that the criteria for body dysmorphic disorder should be broadened to encompass symptoms of both a delusional and a nondelusional nature.

This brings us to the question of how body dysmorphic disorder should be classified (25). Should it be considered a separate diagnostic entity (26), a nonspecific symptom that can occur in a variety of psychiatric syndromes (6), or either (7, 27)? Our impression is that it merits separate classification, given its unique descriptive features, its chronicity regardless of the fluctuation of accompanying disorders, the fact that many patients considered it their most severe and primary problem, and the finding that it may respond to treatment independent of accompanying disorders.

However, this study provides little support for the current classification of body dysmorphic disorder as a somatoform disorder. Indeed, a question raised in the literature is whether body dysmorphic disorder is instead related to, or a variant of, psychosis, mood disorders, obsessive-compulsive disorder, or social phobia (25). Our findings suggest that it is most closely akin, although not identical, to obsessive-compulsive disorder; the two disorders have similar phenomenologic features (obsessive symptoms and compulsive ritualistic behaviors), age at onset and course, and possible preferential response to the serotonin reuptake inhibitors (24, 28, 29). Comorbidity of the two disorders is also fairly high. However, one possible difference is that although obsessive-compulsive disorder sometimes consists of overvalued ideas or delusions ("obsessive-compulsive psychosis" [16, 30, 31]), body dysmorphic disorder appears to consist more often of

overvalued ideation or delusional thinking and to be associated less often with good insight into the senselessness of the preoccupation.

However, some of our findings suggest that body dysmorphic disorder may be related to mood disorders. These findings include high rates of mood disorders in patients with body dysmorphic disorder and their family members, the rate of the latter being similar to rates in relatives of patients with mood disorders (11) (although the occurrence of mood disorders in relatives might be related to mood disorders, rather than body dysmorphic disorder, in the patients). In addition, body dysmorphic disorder appears to respond preferentially to antidepressant medications, although its apparent poor response to ECT and to antidepressants other than the serotonin reuptake blockers somewhat weakens the evidence for a connection between it and mood disorders. The possible relation of body dysmorphic disorder to obsessive-compulsive disorder seems more compelling, but it is possible that both disorders are members of an extended family of mood disorders recently termed "affective spectrum disorder" (32).

Our finding of high comorbidity of body dysmorphic disorder with social phobia is consistent with the Japanese and Korean classification of body dysmorphic disorder as a subtype of "Taijin-phobia" (17, 18), a disorder resembling social phobia or avoidant personality disorder in DSM-III-R. However, more remains to be learned about both body dysmorphic disorder and social phobia before firm conclusions can be drawn about a possible link between them.

Although many earlier authors considered body dysmorphic disorder to be a prodrome or variant of schizophrenia (5, 7, 19, 20), our data suggest that this is probably not the case. The patients responded poorly to neuroleptics, and none had personal or family histories of schizophrenia (delusions were generally not bizarre, and hallucinations were not prominent). And although body dysmorphic disorder symptoms themselves are sometimes delusional, this finding is compatible with a link with obsessive-compulsive disorder and mood disorders.

This study's conclusions are affected by several methodologic limitations. First, the patients were recruited from a psychiatric population, which may have led to elevated rates of associated personal and familial psychopathology, particularly mood disorders and obsessive-compulsive disorder, and may have resulted in a particularly distressed, impaired, and treatment-resistant group of subjects. Second, interviews were done by nonblind investigators and without a control group. Nonetheless, the rates of associated psychopathology were so striking that they seem unlikely to be purely artifactual. In addition, although the extreme distress and impairment experienced by this group may not be generalizable to all individuals with body dysmorphic disorder, they may well describe a significant number or a meaningful subset of those with the disorder, given the consistency of these features in the study group. However, controlled studies of subjects re-

cruited from the general population are needed to clarify these issues.

In conclusion, body dysmorphic disorder appears to be a chronic disorder of significant morbidity and high comorbidity with other psychiatric disorders, particularly mood and anxiety disorders. Its morbidity is further increased by the pursuit of unnecessary surgical, dermatologic, and dental procedures, which appear to alleviate symptoms rarely and more commonly lead to increased preoccupation and further unsuccessful procedures (33). This study also supports the suggestion (24) that body dysmorphic disorder may respond to serotonin reuptake-blocking antidepressants rather than neuroleptics, even if the symptoms are delusional. Indeed, body dysmorphic disorder may be related to obsessive-compulsive disorder or mood disorders, and it may be the same disorder as the body dysmorphic disorder variant of delusional disorder, somatic type. In addition, this disorder is probably far more common than has been recognized, not only because patients often seek nonpsychiatric treatment but also because they are humiliated by their concerns and often keep them secret.

Finally, these findings must be considered preliminary, requiring confirmation by controlled investigation of the phenomenology, course, associated psychopathology, biology, family history, and response to treatment of body dysmorphic disorder. Although this distressing and impairing disorder has been described in the literature for more than a century, empirical work has just begun.

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Personality Disorder in Patients Infected With HIV: A Controlled Study With Implications for Clinical Care

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***Objective:** Previous studies of psychiatric illness in populations infected with or at risk for HIV have not included systematic evaluation for personality disorders. The authors present the first controlled study of 1) personality disorders in HIV-positive and HIV-negative homosexual men and 2) the impact of personality disorder on coping, social support, and mood in asymptomatic HIV-positive homosexual men. **Method:** The authors studied 58 asymptomatic HIV-positive and 53 HIV-negative homosexual men living outside the high-prevalence epicenters of the AIDS epidemic. Personality disorder was assessed with a clinician-administered interview, the Structured Clinical Interview for DSM-III-R. **Results:** There was a significantly higher prevalence of personality disorder in the HIV-positive (33%) than in the HIV-negative (15%) subjects. In the HIV-positive subjects, those with a personality disorder (compared to those without a personality disorder) showed 1) significantly greater mood disturbance, with higher scores on the Hamilton Rating Scale for Depression, Hamilton Rating Scale for Anxiety, Profile of Mood States Total Mood Dysfunction, and the Beck Hopelessness Scale, 2) greater use of denial and helplessness when coping with the threat of AIDS, and 3) greater social conflict. **Conclusions:** These findings suggest that personality disorder is common in the HIV-positive population. Compared with HIV-infected patients without a personality disorder, patients with a personality disorder may experience greater dysphoria and be more likely to cope with the threat of AIDS in a dysfunctional way. Recognition of the impact of personality disorder on coping with HIV infection is important for comprehensive, sensitive, and effective clinical care.*

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Recent studies have consistently found a high lifetime prevalence of major depression and substance abuse in HIV-infected persons (1–4). However, to our knowledge there have been no reports of evaluation of personality disorder in this population. Personality disorders occur when personality traits are “inflexible, maladaptive, and cause either significant functional impairment or subjective distress” (DSM-III-R). It has been suggested that individuals with a personality disorder are at greater risk for many types of psychiatric morbidity (5). Moreover, patients with per-

sonality disorders may pose special problems in the medical management of nonpsychiatric illness (6, 7).

As part of a longitudinal study of neuropsychiatric aspects of HIV infection, we compared the prevalence of DSM-III-R personality disorder in asymptomatic HIV-seropositive homosexual men with the prevalence in a comparison group of seronegative homosexual men. We further examined the impact of personality disorder in HIV-positive homosexual men because personality disorder potentially may affect how an individual adapts to HIV infection. We hypothesized that those with personality disorders would use less effective coping measures, report more dysphoria and greater social conflict, and be more likely to have other psychiatric illnesses than those without personality disorder.

We report here what we believe is the first systematic study of personality disorder in homosexual men with HIV infection. Because attention to psychosocial issues and psychiatric symptoms is essential for the comprehensive treatment of HIV infection, our findings may be useful for clinicians involved in both the general medical and psychiatric care of HIV-infected individuals.

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TABLE 1. Demographic Characteristics of Asymptomatic HIV-Positive and HIV-Negative Homosexual Men Included in and Excluded From Study of Personality Disorders

Item	Study Subjects					
	All Subjects		HIV-Positive Subjects		Excluded Subjects	
	HIV-Positive (N=58)	HIV-Negative (N=53)	Personality Disorder (N=15)	No Personality Disorder (N=39)	HIV-Positive (N=29)	HIV-Negative (N=68)
Age (years)						
Mean	30	31	29	31	32	31
SD	6	7	6	7	8	6
Education (years)						
Mean	14	16	14	14	15	14
SD	2	2	2	3	3	3
Race						
White						
N	45	48	13	32	26	56
%	78	91	68	82	89	83
Nonwhite						
N	13	5	6	7	3	12
%	22	9	32	18	11	17

METHOD

Subjects

Data for the current cross-sectional analysis were collected in North Carolina as part of an ongoing longitudinal study, the Coping in Health and Illness Project, investigating neuropsychiatric, psychosocial, and psychoimmune aspects of HIV infection. The study was approved by the university school of medicine committee for the protection of human rights, and all subjects provided written informed consent.

We studied a total of 111 subjects: 58 asymptomatic HIV-positive and 53 HIV-negative homosexual men. HIV status was determined by ELISA, and positive status was confirmed by the Western blot test. Subjects were recruited from county health departments, from homosexual organizations, by word of mouth, and through newspaper advertisements. North Carolina has a low prevalence of AIDS (8), and thus the cohort consisted of men living outside the high-prevalence epicenters of the HIV epidemic. Table 1 shows that the HIV-positive and HIV-negative subjects were similar in age and education level. The cohort was predominantly white, with some college education, and a mean age of 30–31 years. HIV-positive subjects had known their HIV status for approximately 1 year (mean=392 days, SD=549).

To obtain the study group, we screened 87 HIV-positive and 121 HIV-negative subjects by a telephone interview. Subjects were excluded if they admitted to less than 10 years of education in order to ensure ability to complete questionnaires. Subjects were also excluded if they admitted to intravenous drug use as a risk factor for exposure to HIV, since we wished to limit the cohort to those individuals with homosexuality as their primary risk factor for HIV. In addition, subjects were excluded if they admitted to 1) age less than 18 or more than 50 years, 2) significant medical illness, 3) history of heavy alcohol or drug use, and 4) zidovudine use.

These criteria were needed in order to study neuropsychiatric and psychoimmune relationships. Table 1 shows that excluded subjects were similar to the study subjects in age, race, and educational status.

We used the results of the initial screening evaluation to determine whether a subject would be included in the study. However, following their complete study evaluation, some subjects included in this analysis were found to actually meet one of these exclusion criteria. Specifically, 11 HIV-positive and four HIV-negative subjects had a history of heavy alcohol or drug use, and six HIV-positive subjects had a history of zidovudine use.

Procedure

All subjects were evaluated in a university general clinical research center. The 2-day inpatient evaluation included physical, neurological, and neuropsychological examinations and life stress and psychiatric interviews. On the first evening of their admission subjects completed a 348-item questionnaire that assessed mood, psychosocial behavior, and health habits.

DSM-III-R axis I and axis II disorders were assessed by a trained psychiatric clinician with a modified Structured Clinical Interview for DSM-III-R (SCID) (9, 10) and the Structured Clinical Interview for DSM-III-R Personality Disorders (SCID-II) (11), respectively. For the SCID-II, subjects first completed a 113-item questionnaire that explored symptoms of personality disorders. The interviewer then reviewed the questionnaire with the subject, probing each item answered affirmatively to determine if the subject met criteria for the particular personality disorder trait. The interviewer was not limited only to items endorsed by the subject and probed any other areas considered important. A subject was considered to have personality disorder traits if he had symptoms in a particular area that caused significant disturbance in functioning, without meeting the full criteria for the personality disorder. A

subject received the diagnosis of "not otherwise specified" if he had symptoms from more than one personality disorder that caused significant impairment in functioning but did not meet criteria for a specific personality disorder (DSM-III-R). In subjects who had a diagnosis of multiple personality disorder, the principal personality disorder diagnosis was defined as the personality disorder that should be the main focus of clinical attention (11).

Diagnoses were assigned at a diagnostic conference after review of clinical material. A videotape format was used to assess interrater reliability. Interrater reliability for determination of a DSM-III-R personality disorder was good, with a kappa coefficient of 0.67. Reliability for assessment of the specific personality disorders could not be assessed because of small group size. Interrater reliability for axis I disorders was good to excellent, with kappa ranging from 0.75 to 0.92.

Trained psychiatric clinicians evaluated severity of dysphoric mood with the Hamilton Rating Scale for Depression (12) and the Hamilton Rating Scale for Anxiety (13). Interrater reliability was excellent, with intraclass correlation coefficients of 0.99 for the Hamilton depression scale and 0.96 for the Hamilton anxiety scale. Subjects also completed the Profile of Mood States (POMS), previously shown to have good reliability and validity (14). We used the anger, tension, depression, and total mood disturbance scales of the POMS in this study. We evaluated hopelessness with the Beck Hopelessness Scale (15). The Carroll Rating Scale for Depression (16) was used as a self-report measure of syndromal depression.

We determined the number of social supports and satisfaction with available social supports by using the previously validated Sarason's Brief Social Support Questionnaire (17). Social conflict was measured by a 7-item inventory used in the Chicago Multicenter AIDS Cohort Study in the Coping and Change Survey (unpublished 1991 manuscript of K. O'Brien et al.). Social conflict examines the level of discord during the past month in close personal relationships. Items included how frequently "people in their life let [them] down," had "gotten on [their] nerves," and had made the subject "feel respected" or "feel tense from arguing"; the frequency of interactions that made the subject "feel irritated or resentful" or "feel misunderstood"; and the frequency of unpleasant social interactions. Response categories ranged from 1 ("no, never") to 5 ("yes, all the time") and were summed and divided by seven to obtain the scale score.

Coping was assessed with the use of a modified version of the COPE (18). Subjects were asked to indicate on a 4-point scale (ranging from "not at all" to "very much") how much they "generally cope with or handle the threat of getting AIDS." Seven of the 14 COPE scales were used in this analysis, including seeking social support, positive reinterpretation and growth, planning, turning to religion, denial, fighting spirit, and helpless coping.

Reliability for self-report measures was substantial,

with Cronbach's alpha as follows: POMS anger=0.88, POMS tension=0.90, POMS depression=0.90, Sarason's social support number=0.90, Sarason's social support satisfaction=0.92, Social Conflict Scale=0.88, and COPE scales=0.80 to 0.93.

Statistical Analysis

To compare the prevalence of personality disorder in HIV-positive and HIV-negative individuals we used logistic regression. HIV status was considered the dependent variable. We controlled for age, race, and years of education.

To determine the relationship of mood, social support, and coping variables with personality disorder, we used analysis of variance (ANOVA)/analysis of covariance. For each model, presence or absence of personality disorder was the independent variable. Covariates included age, race, years of education, zidovudine use, helper cell (CD4+) count, and length of time the subject had known his HIV status (truncated at 1 year to avoid outlier effects). None of the covariates was significant. Thus, we report unadjusted means. Furthermore, none of the control variables altered the relationship between personality disorder and mood, coping, and social support.

We used the chi-square test and Fisher's exact test (when cell sizes were small) for simple comparisons of proportions. Data were analyzed using the Statistical Analysis Software package. All reported p values are for two-tailed tests of significance.

RESULTS

Prevalence of Personality Disorder

There were significantly more individuals with DSM-III-R personality disorder among HIV-positive subjects (33%) than among HIV-negative subjects (15%) ($\chi^2=4.7$, $df=1$, $p=0.03$). Controlling for race and age did not alter this finding. However, controlling for education reduced the finding to a trend ($\chi^2=2.4$, $df=1$, $p=0.11$).

For the 19 HIV-positive subjects with personality disorder diagnoses, 13 received a single personality disorder diagnosis, and six received more than one such diagnosis. For the eight HIV-negative subjects, five received a single personality disorder diagnosis, and three received more than one diagnosis. The most common principal personality disorder diagnosis (except for not otherwise specified) was borderline personality disorder (five HIV-positive subjects, three HIV-negative subjects). Other principal personality disorder diagnoses included dependent (one HIV-positive subject), passive-aggressive (one HIV-negative subject) histrionic (three HIV-positive subjects, one HIV-negative subject), and not otherwise specified (10 HIV-positive subjects, three HIV-negative subjects). For the 13 individuals who received a diagnosis of personality disorder not otherwise specified, the majority (six HIV-positive

TABLE 2. Frequency of DSM-III-R Axis I Disorders and HIV Status of Homosexual Men With and Without Personality Disorder

Axis I Disorder	HIV-Positive Men					HIV-Negative Men					p (Fisher's exact test)	p (Fisher's exact test)
	Personality Disorder				p (Fisher's exact test)	Personality Disorder				p (Fisher's exact test)		
	Yes (N=19)		No (N=39)			Yes (N=8)		No (N=45)				
	N	%	N	%		N	%	N	%			
Major depression												
Current	3	16	3	8	0.38	0	0	1	2	1.00	0.12	
Lifetime	10	53	14	36	0.27	3	38	18	40	1.00	1.00	
Current dysthymic disorder	1	5	1	3	1.00		13	1	2	0.28	1.00	
Anxiety disorder												
Current	0	0	0	0	—		13	0	0	0.15	0.48	
Lifetime	0	0	0	0	—		13	1	2	0.28	0.23	
Eating disorder												
Current	0	0	0	0	—		0	0	0	—	—	
Lifetime	1	5	1	3	1.00		0	0	0	—	0.50	
Alcohol dependence												
Current	1	5	5	13	0.65		0	1	2	1.00	0.12	
Lifetime	4	21	10	26	1.00		63	8	18	0.02	1.00	
Drug dependence												
Current	0	0	0	0	—		0	0	0	—	—	
Lifetime	4	21	5	13	0.46		38	6	13	0.12	1.00	
Any axis I disorder												
Current	5	26	8	21	0.74		25	3	7	0.16	0.08	
Lifetime	16	84	28	72	0.35		75	32	71	1.00	0.67	

subjects, two HIV-negative subjects) had borderline traits, along with a variety of other personality disorder traits needed to give the diagnosis of not otherwise specified.

Comorbidity With Axis I Disorders

Table 2 shows the proportion of lifetime and current (in the past month) axis I disorders in subjects with and without a personality disorder, by HIV status. In the HIV-negative group, lifetime alcohol dependence occurred in a significantly greater proportion of the subjects with a personality disorder than in the subjects without a personality disorder. Otherwise, no significant differences were found. Table 2 further shows that there were no significant differences in the overall proportion of axis I disorders in HIV-positive and HIV-negative individuals.

Impact of Personality Disorder on Coping With HIV Infection

To investigate the impact of personality disorder on coping with HIV infection, we examined the relationship between presence of personality disorder and mood, coping, and social support in the 58 HIV-seropositive subjects. We considered the presence or absence of any personality disorder because of multiple personality diagnoses in some individuals and relatively low frequency of specific DSM-III-R personality disorders. In addition, while personality disorder may be overdiagnosed in individuals who are currently depressed (19, 20), only six subjects had a current major depression, and there was no significant relationship

between a current major depression and personality disorder ($\chi^2=0.9$, $df=1$, $p=0.30$). Thus, major depression was not considered a potential confounder for further analyses.

Current Mood Disturbance

As shown in table 3, we found significantly greater levels of mood disturbance, including depression, anxiety, anger, tension, and hopelessness, among the subjects with a personality disorder. We show the unadjusted means for the mood variables because the covariates age, education, race, length of time a subject had known his HIV status, CD4+ count, and zidovudine use were not significant.

Coping

Table 4 shows the relationship between personality disorder and coping with the threat of AIDS. We show the unadjusted means for the coping variables because the covariates age, education, race, length of time a subject had known his HIV status, CD4+ count, and zidovudine use were not significant.

Individuals with a personality disorder showed significantly greater use of denial and helpless coping styles than subjects without a personality disorder. Denial is exemplified by the following beliefs: "I refuse to believe it has happened, I pretend it hasn't really happened." People who use helpless coping endorse such statements as, "I feel that there is nothing I can do to help myself, I am unable to handle the stress of AIDS." There was no significant relationship between personality disorder and the other coping styles in the subjects.

TABLE 3. Mood Disturbance Scores of Asymptomatic HIV-Positive Homosexual Men With and Without Personality Disorder

Measure	Subjects' Scores				ANOVA		
	Personality Disorder (N=19)		No Personality Disorder (N=39)		F	df	p
	Mean ^a	SD	Mean ^a	SD			
Hamilton scale							
Anxiety ^b	6.4	5.5	3.4	3.8	4.7	1, 48	0.03
Depression ^c	7.9	5.8	3.6	3.8	10.5	1, 52	0.002
Carroll depression scale	13.3	6.5	7.3	6.1	11.6	1, 56	0.001
POMS							
Total ^d	41.2	38.7	22.9	23.9	4.8	1, 55	0.03
Anger ^d	11.4	9.0	7.3	4.7	5.0	1, 55	0.03
Tension ^d	14.9	8.2	10.1	4.9	7.6	1, 55	0.008
Depression ^d	15.9	11.0	8.6	6.4	10.1	1, 55	0.003
Beck Hopelessness Scale	6.8	5.0	3.2	3.7	9.7	1, 56	0.003

^aUnadjusted means are reported because covariates of age, race, years of education, CD4+ count, zidovudine use, and length of time that subject knew HIV status were not significant.

^bHIV-positive subjects, N=13; HIV-negative subjects, N=37.

^cHIV-positive subjects, N=15.

^dHIV-positive subjects, N=18.

TABLE 4. Coping Scores of Asymptomatic HIV-Positive Homosexual Men With and Without Personality Disorder

Coping Style ^a	Subjects' Scores				ANOVA		
	Personality Disorder (N=19)		No Personality Disorder (N=39)		F	df	p
	Mean ^b	SD	Mean ^b	SD			
Fighting spirit	3.4	0.5	3.3	0.4	0.2	1, 56	0.64
Growth	3.1	0.6	3.0	0.6	0.3	1, 56	0.56
Planning	3.0	0.9	3.0	0.7	0.02	1, 56	0.89
Seek social support	2.7	0.9	2.8	0.7	0.4	1, 56	0.52
Religion	2.9	0.7	2.5	1.0	2.1	1, 56	0.16
Denial	2.1	0.8	1.4	0.4	18.5	1, 56	0.0001
Helpless	1.9	0.5	1.5	0.4	10.6	1, 56	0.002

^aThe coping scales ranged from 1 (low) to 4 (high).

^bUnadjusted means are reported because covariates of age, race, years of education, CD4+ count, zidovudine use, and length of time that subject knew HIV status were not significant.

Social Support and Conflict

Subjects with a personality disorder reported significantly more social conflict than those subjects without personality disorder, with none of the covariates significant ($F=11.4$, $df=1, 56$, $p=0.001$). We did not find differences in the number of social supports ($F=0.3$, $df=1, 56$, $p=0.60$) or satisfaction with social supports ($F=1.2$, $df=1, 55$, $p=0.27$) in subjects with personality disorder.

CONCLUSIONS

Some data on axis I disorders are now available for populations at risk for AIDS (1-4), but to our knowledge this is the first report on axis II disorders in individuals with HIV infection. Personality disorder was comparatively frequent in our group of HIV-infected homosexual men, with 33% receiving this diagnosis. The prevalence of personality disorder in the general population has been estimated to be between 5% and

15% (5). However, comparisons with our study are problematic because the studies from which these rates were derived did not use DSM-III-R criteria. If our findings are confirmed, a possible explanation for a high proportion of HIV-infected men having personality disorder may be that individuals with a personality disorder are more likely to engage in high-risk sexual behavior, leading to HIV exposure. It should be emphasized that a causal connection between personality disorder and high-risk sexual behavior has *not* been shown by this cross-sectional study. However, our explanation for the finding of greater prevalence of personality disorder in HIV-positive individuals is plausible, given that individuals with a personality disorder, especially those with borderline features, commonly engage in impulsive and self-destructive behavior (5).

People with personality disorders have often been regarded as causing more distress to those around them than they experience themselves (5). While we do not have a measure of the distress that our subjects produced in others, we do have evidence that in this HIV-seropositive group, individuals with a personality dis-

order suffered significantly more emotional distress and conflict with others than those without a personality disorder. There was no excess of individuals with a personality disorder with axis I diagnoses, but on all measures of current mood disturbance there was a significantly greater degree of dysphoria in the presence of a personality disorder. These findings extend to the HIV-infected homosexual population the recent and growing clinical appreciation that individuals with personality disorder suffer considerably more dysphoria than control subjects (21).

Dysphoric mood, denial, and helplessness reported by the subjects with a personality disorder may be interrelated. A tendency to deny or feel helpless when considering the threat of AIDS may underlie the emotional distress seen in the individuals with a personality disorder. These coping strategies may hinder patients from accepting and dealing with their HIV infection. Alternatively, individuals with personality disorders may experience greater dysphoria, affecting their ability to cope with the threat of AIDS. Regardless, the greater use of denial and helpless coping potentially may affect the relationship between the patient and the medical care provider and ultimately compliance with medical treatment. Psychiatric intervention may be of value in helping the patient better cope with his illness, particularly when the relationship between care provider and patient is compromised.

It is not surprising that our subjects with personality disorder experienced more conflict with the people in their personal lives than did subjects without a personality disorder. However, it is interesting that this conflict did not affect the number of social supports or the individual's satisfaction with his social support. This finding may reflect a relative adaptation of others to the personality function of the disordered individual or the impaired ability of those with a personality disorder to assess their social relationships.

The results of this study suggest that DSM-III-R axis II personality disorders may occur frequently in HIV-infected asymptomatic homosexual men. There was no difference in the prevalence of axis I disorders between the seropositive subjects with and without personality disorders in this cross-sectional assessment; however, subjects with a personality disorder who go on to develop an axis I disorder may respond less well to psychiatric intervention (22). Thus, it is important for mental health professionals to assess both axis I and axis II disorders in their HIV-seropositive patients. Moreover, findings from the present study suggest that HIV-infected homosexual men who meet criteria for a personality disorder may experience greater dysphoria and poorer coping styles. Because the personality disorder diagnoses in the present study were mainly borderline and not otherwise specified with borderline and other traits, it may be that these findings pertain only to individuals with these diagnoses and not to others.

There are some important caveats to consider in evaluating these results. Similar to other studies that

depend on a volunteer cohort, the generalizability of our study findings is limited in that the subjects were all self-selected homosexual men and may differ systematically from homosexual men who did not volunteer to participate. In particular, most of the subjects were well educated and white. It is important to note that it is not feasible to obtain a random sample of our target populations (HIV-infected and uninfected men with homosexuality as their HIV risk factor), since members of the target population cannot be systematically determined. Subjects were also required to meet a variety of inclusion criteria, further restricting our cohort and thus limiting generalizability. In particular, our subjects and homosexual men who did not volunteer or meet inclusion criteria may differ systematically on the prevalence and type of personality disorder. Thus, our study prevalence of personality disorder may actually be an underestimate, since we excluded persons with a history of heavy substance use, a condition often associated with many personality disorders (23). However, comparisons between HIV-positive and HIV-negative groups and within HIV-positive subjects are valid because recruitment and screening were done similarly with all subjects.

Group size limits the interpretation of negative findings. Although we studied 58 HIV-positive subjects, 19 with a personality disorder, a larger cohort might demonstrate a difference in some other measures that occurred infrequently, such as the prevalence of current or lifetime psychiatric disorders. The group size also limited the evaluation of specific personality disorders. It is interesting that the significant difference in prevalence of personality disorder between the HIV-positive and HIV-negative groups was reduced to a trend when education was taken into account. This may speak to the functional impairment of individuals with personality disorders, which leads to inability to remain in school as long as their peers without personality disorders.

Comprehensive treatment of individuals infected with HIV requires an understanding of psychiatric and psychosocial factors (4). Our results are important for both psychiatric and nonpsychiatric clinicians treating individuals with HIV infection, who need to be aware of potential psychiatric illnesses, including personality disorders, in these patients. Furthermore, since the subjects did not reside in an epicenter for AIDS, they may reflect the HIV epidemic as it currently exists and will exist in the future for most of the United States. We hope that the findings of the present study, which demonstrate a relatively high prevalence of personality disorder in HIV-infected asymptomatic homosexual men, will encourage other researchers to investigate the importance of personality disorder in their population of HIV-infected individuals. We also hope that our findings will raise the awareness of both psychiatric and nonpsychiatric clinicians for the existence of personality disorder in HIV-infected patients in order that they may provide comprehensive, sensitive, and effective clinical care.

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Quality Assessment and Improvement in Group Psychotherapy

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***Objective:** The authors sought a practical means of monitoring and evaluating group psychotherapy, using existing clinical resources, for purposes of quality improvement and education on a large general hospital psychiatric service. **Method:** Monitoring indicators were developed which addressed 1) the integration of group psychotherapy into treatment planning and 2) the competence and technique of group psychotherapists. The second indicator was assessed by skilled observers using a newly constructed Group Psychotherapy Rating Scale in direct observation of group psychotherapy sessions. The rating scale was examined for interrater reliability and, as a measure of construct validity, for its ability to distinguish the performance of professional staff therapists from that of trainees. **Results:** The indicators provided useful monitors of the use and quality of group psychotherapy. The rating scale had satisfactory interrater reliability and construct validity. The immediate constructive educational critique given by the observers of the therapy groups was highly valued by group therapists. **Conclusions:** The monitoring and evaluation program proved to be a practical, positive, and inexpensive means of assuring and improving the quality of group psychotherapy in a clinical setting.*

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The construction of a quality assessment and improvement program in a health care facility usually follows the 10 steps recommended by the Joint Commission on Accreditation of Healthcare Organizations (JCAHO), among which are the identification of major aspects of care in the facility and the selection of indicators to monitor those aspects of care (1). Major aspects of care such as performance of appendectomies on a surgical service or treatment of infections on a medical service lead readily to quantifiable indicators (e.g., the proportion of normal appendixes removed or the incidence of infections treated without obtaining cultures). However, the therapeutic interventions of psychiatric services or facilities have been less amenable to the development of similar quantitative indicators in some important kinds of treatment. Psychotherapy of various types, for example, is clearly a major aspect of care in most psychiatric programs, yet it is not easily accessible to conventional methods of measurement or evaluation in a reproducible fashion. Although psychotherapy has been observed and evaluated as part of scientifically rigorous research to determine its clinical effectiveness (2) or as a means of supervising trainees in

academic programs (3), the published literature contains no accounts of monitoring psychotherapy as a means of quality assessment and improvement (or, in older terms, quality assurance). There are, for example, no indicators addressing psychotherapy among those suggested in a recent review of quality assurance in psychiatry (4), a JCAHO publication on the quality assurance process (1), and a quality assurance guide published by an association of private psychiatric hospitals (5).

At our institution we identified group psychotherapy as a major aspect of care that was utilized throughout our large inpatient and outpatient psychiatry service and conducted by staff members and trainees from several health care disciplines; there were 65 active therapy groups on inpatient and outpatient units. We therefore set out to construct a multidisciplinary group psychotherapy monitoring program as a quality assessment measure and as an educational tool for quality improvement. In this article we describe the development process and report the results of the first two monitoring periods.

METHOD

As a first step, the psychiatry service administration formed a multidisciplinary team—consisting of a psychiatrist (J.S.), a clinical psychologist (P.H.), a social worker (T.J.C.), and a registered nurse (C.S.)—to perform the monitoring and education tasks. All members

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of the team were senior staff members with formal professional training in group psychotherapy and at least 5 years' experience in leading psychotherapy groups. The team surveyed the psychiatry service to determine the number, locations, orientation, and leadership of the active psychotherapy groups. The team then devised two broad indicators to monitor group psychotherapy: an "external" indicator to assess whether group psychotherapy, when prescribed within a unit's treatment program, was integrated into the overall planning and monitoring of treatment, and an "internal" indicator to assess whether group psychotherapy sessions were conducted with professionally accepted techniques and competence.

To define operationally the criteria for the first indicator, the team composed a worksheet (copies are available from the first author on request) which recorded, for each psychiatry service unit, whether a written policy regarding patients' participation in therapy groups was present and implemented, whether group therapists regularly shared relevant information from the therapy groups during treatment planning conferences and in the clinical record, and whether group therapists were provided supervision or consultation on a scheduled basis. Team members completed these worksheets for all psychiatric units by interviewing unit staff members and observing treatment planning conferences.

To define the criteria for the second indicator, the team constructed the Houston VA Medical Center Group Psychotherapy Rating Scale (copies are available from the first author on request), an evaluation instrument that included six criteria for group process judged to be common to all theories of group psychotherapy. In the initial version, six additional criteria more germane to psychodynamic and interpersonal theories were included for educational purposes only and were excluded from the formal quality assessment. Each of the criteria was graded on a 7-point scale, with anchoring descriptions constructed so that a score of 4 or more was considered satisfactory.

This instrument was refined by pilot testing and by soliciting suggestions from psychiatric unit directors and experienced group psychotherapy leaders. The team then performed an interrater reliability study in which each of the six possible pairs of team members observed a group together, and each member of the pair independently completed the rating scale. Reliability was then measured by two methods. First, as an unweighted measure, a Pearson product-moment correlation coefficient was calculated for each pair of raters across all items. Second, as a measure that considered the differing magnitudes of disagreement, a weighted kappa (6) was calculated for each pair of raters across all items; the disagreement weight for each cell was set at $(i-j)^2$, where i and j were the respective ratings by the two raters.

Before initiating formal monitoring, the team met collectively with all psychotherapy group leaders to explain the intent and procedures of the monitoring program. The team emphasized the educational and qual-

ity improvement goals of the program and assured the staff that only aggregate results of monitoring would be published in psychiatry service quality assessment reports. Results for individual therapists were to be shared only with their respective professional disciplines' service. The team distributed the rating scale and encouraged discussion of staff members' questions and concerns.

For the first 6-month period of observation, a sample of 16 of the 65 identified therapy groups was selected to include at least one group from each psychiatry service unit and to maximize the number of different health care disciplines of the group leaders. Educational, orientation, and exercise groups were excluded from this selection. Each of the 16 selected groups was observed twice during the 6-month period by a single team member. Team members were arbitrarily assigned as observers without regard to the professional discipline of the observer relative to the discipline of the group therapists.

The therapists in each group selected were informed of the selection and of the two dates on which the observer would be present. The therapists were also given written guidelines on preparing their group members for the arrival of the observer. The observer entered the therapy room a few minutes in advance of a session to position himself or herself outside the group if possible. After introductions, the observer did not respond to any of the group's interaction. If more than one therapist was present in the group, the observer performed separate ratings of each therapist. Immediately following the group session, the observer and the therapists reviewed the results of the rating scale. The observer also offered suggestions about therapy techniques and alternatives for handling any critical incidents observed during the group session. Any group therapist failing to meet the established criteria was offered in-service training by the team.

A detailed report of findings, conclusions, and recommendations was submitted to the psychiatry service's quality assurance committee. Specific data on therapists were provided to psychiatry, psychology, social work, and nursing services in separate reports.

After the first 6-month period, the monitoring team revised the Group Psychotherapy Rating Scale to improve the observers' ability to distinguish satisfactory from highly skilled group work. In the final version of the rating scale, anchor point definitions were revised, and two of the education-only items were dropped because they had not been found useful educationally. Fourteen new groups, none of which had been included in the first sample, were then observed and rated twice during the next 6-month period with the same protocol, except for these minor modifications.

Ratings from each of the two periods were compared by means of descriptive statistics, and a two-way analysis of variance (ANOVA) for unequal numbers of subjects was performed on the sums of the ratings of the six required items for the professional staff therapists and for trainees in both rating periods. As a test of con-

TABLE 1. Compliance of 10 Hospital Psychiatric Units With Indicator 1 (Integration of Group Psychotherapy Into Treatment Planning) for Monitoring Use and Quality of Group Psychotherapy

Indicator Criterion Item	Compliance (%)	
	Period 1	Period 2
Unit has written policy and procedures for patient participation in group psychotherapy	40	80
Unit policy is implemented	40	70
Group psychotherapy is documented as part of the treatment plan	80	100
Patient's treatment plan designates specific therapy groups	60	70
Group therapists give feedback on patient's progress at treatment staffing conference	70	90
Unit has written policy concerning frequency of chart notes to document course of group psychotherapy	20	60
Cotherapists, if present in a group, meet before or after group sessions to discuss group issues	70	100
All group therapists on the unit meet regularly to discuss group issues	40	60
Group therapists receive regular supervision or consultation	20	40

struct validity, we hypothesized that 1) ratings in the second period would be lower than those in the first period because of our efforts to "tighten" the rating scale and 2) ratings of the professional staff therapists would be higher than those of the trainees.

RESULTS

Table 1 shows the compliance with the criteria for the first indicator, during successive 6-month periods, regarding the integration of group psychotherapy into units' overall planning and monitoring of treatment. Progressive improvement in compliance with all criteria is evident, although some units did not reach full compliance until after the monitoring periods reported here.

Results of the reliability study for the Group Psychotherapy Rating Scale are shown in table 2. There was satisfactory to excellent correlation between raters, with a mean of 0.82. The weighted kappa statistics, ranging from 0.44 to 0.90, also demonstrated fair to excellent agreement beyond chance, according to the usual standards of interpretation of the kappa statistic (7).

Table 3 shows the items of the Group Psychotherapy Rating Scale and the mean and range of ratings for each item during the two 6-month rating periods. In the second rating period, most items were rated with a lower mean and a wider range, consistent with the goal of the scale revision to distinguish better between satisfactory and highly skilled group therapists' performance. As confirmation, the ANOVA showed a significant main effect for rating period ($F=8.86$, $df=1$, 60 , $p=0.004$). Of the two exceptions in which ratings rose rather than fell in the second period, the rise for the item measuring whether cotherapists work well as a team may have re-

TABLE 2. Interrater Reliability on the Group Psychotherapy Rating Scale of Observers Monitoring Use and Quality of Group Psychotherapy in 10 Hospital Psychiatric Units

Rater Pair	Absolute Difference on Items Rated		Pearson r	Weighted kappa
	Mean	SD		
AB	0.90	0.99	0.87	0.64
AC	1.00	0.82	0.72	0.44
AD	0.56	0.53	0.90	0.90
BC	0.70	0.48	0.99	0.90
BD	0.33	0.49	0.57	0.57
CD	0.50	0.52	0.89	0.85
Mean	0.65	0.68	0.82	0.72

flected the increasing competence of junior cotherapists (usually trainees) in the second half of the academic year, which roughly coincided with the second rating period; however, the overall interaction of status and period was not significant ($F=0.14$, $df=1$, 60 , $p=0.71$). The rise in the rating for the item measuring in-depth work with group members' feelings is discussed in the following section.

The ANOVA also demonstrated the rating scale's significant discrimination between the performance of the professional staff (including psychiatrists, psychologists, and social workers) and that of the trainees (including psychiatric residents, psychology interns, and social work students) on the six required criterion items (main effect for status, $F=4.07$, $df=1$, 60 , $p=0.05$). This finding was in the expected direction: the mean total score on the rating scale was 35.6 (range=25–42) for the professional therapists and 33.6 (range=22–40) for the trainees.

In the first rating period, all group therapists received ratings of 4 (the predefined threshold for satisfactory performance) or more on all of the six required criterion items of the Group Psychotherapy Rating Scale, except for two students acting as temporary cotherapists in two different groups. In the second rating period, one group therapist received unsatisfactory scores on two required items during the first observation; after corrective educational measures were implemented, this therapist received excellent scores for all items during the second observation (by the same rater). Another group therapist showed deficiencies on two items during the second rating period, but the group in question was discontinued for reasons unrelated to the rating.

DISCUSSION

Our results indicate that our method of monitoring group psychotherapy was generally successful as a quality assessment and improvement measure. We constructed and implemented process indicators, as opposed to outcome indicators, because in our clinical setting it was not possible to isolate in experimental fashion the outcome effect of group therapy from the

TABLE 3. Compliance of Therapists in 10 Hospital Psychiatric Units With Indicator 2 (Group Psychotherapy Technique) for Monitoring Use and Quality of Group Psychotherapy

Indicator Criterion Item ^a	Rating for Item			
	Period 1		Period 2	
	Mean	Range	Mean	Range
Required				
Therapist listens to and hears each group member out and checks for understanding of what is being said	6.1	5-7	5.9	4-7
Therapist gives feedback to group members about their behavior	6.0	4-7	5.3	2-7
Therapist facilitates interactions between or among group members	5.7	3-7	5.5	1-7
Therapist is sensitive to group members' level of participation	5.6	3-7	5.2	2-7
Therapist facilitates supportive group atmosphere	6.4	5-7	6.1	4-7
If more than one therapist, therapists work well as team	5.5	5-7	5.7	4-6
For educational discussion only				
Therapist shows awareness of group process by making group-level observations	4.8	1-7	3.9	1-6
Therapist focuses on "here and now" dynamics within group	6.2	3-7	4.5	1-7
Therapist attends to nonverbal cues	4.6	1-7	3.7	1-7
Therapist works in depth with group members' feelings	3.4	1-6	5.3	2-7

^aEach item was rated on an anchored 7-point scale, with a score of 4 or more defined as satisfactory.

many other components of each patient's treatment; no patient received group therapy as the sole treatment intervention. We believed that ensuring high quality in the process of conducting group therapy and integrating the group work into the total treatment plan would tend to produce a good outcome. Research in group therapy supports the relation of process measures to outcome (8, 9). In particular, Dies and Teleska (10) have noted that certain styles and actions by group therapists impede the curative factors of group therapy (11) and correlate with negative outcome.

An important feature of our monitoring system is the use of two different kinds of indicators. The first indicator, regarding the integration of group therapy into treatment planning, consists of very concrete criteria whose fulfillment or nonfulfillment is susceptible to objective, reproducible identification and can be ascertained for the most part from written documents. This type of indicator is common in quality assessment programs because acquisition of the necessary data is relatively simple and does not require highly skilled personnel. Nonetheless, we felt that such an "external" indicator, while useful in its own right, did not address the core of group psychotherapy—namely, what was actually occurring during the therapy sessions. For this reason we developed our "internal" indicator, which involves direct observation of group therapists in actual clinical practice. Despite requiring a much higher level of inference and expert knowledge, ratings for this indicator showed a satisfactory level of interrater agreement and functioned as desired in terms of the scoring range used and the correlation with level of therapist training.

Alternative methods of observing groups for rating the items of the second indicator might have been the use of a one-way mirror or a videotape of the group session, thereby removing the observer from the room. Such methods might have lessened the possible effects of the observer's presence on the therapists' performance and the possible discomfort of group members at

being observed (12). However, these methods have disadvantages as well: patients and therapists are still aware of being observed, and a video camera, because of its relatively fixed location, will miss certain behaviors and nonverbal communications in the group. In any case, such alternatives were not logistically practicable in our situation of monitoring a large number of groups in multiple locations. We did attempt to minimize the observers' effect on group therapists and group members by the extensive preparation described in the Method section, which appeared to produce a high degree of acceptance of the observation process. Indeed, observers noted that once they were introduced and their purpose restated, the group members seemed to proceed as usual. Group therapists reported no significant negative effects and little or no time required in subsequent sessions to deal with the residual effects of observation of the group.

The educational aspect of our monitoring process was generally well received by group therapists, many of whom expressed appreciation for the immediate, constructive critique as well as for the availability of additional in-service training. Some possible evidence for the impact of the educational component was the large increase in the rating on the item "Therapist works in depth with group members' feelings" during the second rating period. We speculate that this increase may have resulted from observers' emphasizing this aspect of group therapy in their feedback to therapists during the first rating period and consequent discussion about it among group therapists. Further positive effects included one unit's requesting a series of educational sessions from the monitoring team for all the unit staff involved in group work and another unit staff's decision to observe one another as a means of improving their group therapy through peer feedback. We believe that an educational component of this sort is crucial to ensure that the monitoring is seen by staff not merely as a critical evaluation of performance but also as a positive means for continuous quality improvement.

A final noteworthy aspect of our monitoring is its cost efficiency. During the first 6 months, when the instruments were being developed, implemented, and refined, each of the four team members spent only 48 minutes per week on this activity. During the second 6 months, each spent only 20 minutes per week. Even with a generous cost estimate of \$100 per hour of staff time, the cost of the monitoring was about \$80 a week per team member during start-up and only about \$33 a week per member in subsequent periods. Costs in other institutions, where earnings may be more sensitive to hours of patient contact, can be lessened further by using our already-developed instruments and having fewer team members. Costs will also be lower if there are fewer groups to monitor. Thus, as our experience demonstrates, the innovative use of existing resources can cost-effectively enhance both the quality of care and the education of the caregivers.

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A Double-Blind Crossover Pilot Study of *l*-Deprenyl (Selegiline) Combined With Cholinesterase Inhibitor in Alzheimer's Disease

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The potential efficacy of oral l-deprenyl (5 mg b.i.d.) added to the regimen of 10 patients with Alzheimer's disease receiving either tacrine or physostigmine was assessed in a double-blind, placebo-controlled, 4-week, two-period crossover pilot study. l-Deprenyl was associated with significant improvement in scores on the cognitive subscale of the Alzheimer's Disease Assessment Scale, suggesting possible additive effects of l-deprenyl to the effects of cholinesterase inhibitors.

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The drug *l*-deprenyl is a centrally and peripherally acting irreversible monoamine oxidase inhibitor (MAOI) that in low doses (10 mg/day) selectively inhibits MAO-B. It has been reported to improve agitation, anxiety, episodic learning, and memory in some patients with Alzheimer's disease (1) and may lower the rate of deterioration in Parkinson's disease, consistent with a putative neuroprotective effect (1). MAO-B activity in patients with Alzheimer's disease is greater than the elevated activity associated with aging alone (2). Differences in platelet MAO activity between groups of patients with Alzheimer's disease may be specific to such symptomatic behaviors as depression or agitation and delusions (3).

Substantial evidence now exists that cholinesterase inhibitors such as tacrine and physostigmine exert modest but clinically significant cognitive effects in patients with Alzheimer's disease (4, 5). Interestingly, ani-

mal studies suggest that intact monoaminergic systems facilitate the effect of cholinergic medications (6). Therefore, we undertook a pilot trial of low doses of *l*-deprenyl to assess its potential cognitive effects in patients with Alzheimer's disease who were receiving a cholinesterase inhibitor. We hypothesized that patients with Alzheimer's disease would show improvement in cognitive function as assessed by scores on a standard instrument used in dementia trials.

METHOD

Study Design

Patients who were already receiving tacrine or sustained-release physostigmine salicylate were randomly assigned under double-blind conditions to receive either a 4-week trial of oral *l*-deprenyl (5 mg b.i.d.) followed by 4 weeks of placebo or a 4-week trial of placebo followed by 4 weeks of *l*-deprenyl administration, without a washout period between trials. (Medication and placebo were dispensed as identical-appearing tablets.)

The subjects were outpatients with probable Alzheimer's disease who had participated in and completed multicenter clinical trials of tacrine or sustained-release physostigmine (4) and had been receiving the cholinesterase inhibitor since the trial ended. Briefly, inclusion criteria for the trials (detailed elsewhere [4]) were age

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TABLE 1. Scores on the Mini-Mental State Examination and the Cognitive Subscale of the Alzheimer's Disease Assessment Scale of 10 Patients With Alzheimer's Disease Treated With Tacrine or Physostigmine Plus *l*-Deprenyl

Subject	Age (years) ^a	Education (years) ^b	Sex	Concurrent Medication	Dose of <i>l</i> -Deprenyl	Mini-Mental State Examination Score ^c		Cognitive Subscale Score ^d	
						Placebo	<i>l</i> -Deprenyl ^e	Placebo	<i>l</i> -Deprenyl ^e
1	63	13	F	Physostigmine (30 weeks)	15 mg/day	16	12	29	26
2	60	14	M	Tacrine (52 weeks)	10 mg q.i.d.	16	14	40	32
3	71	14	M	Tacrine (32 weeks)	10 mg q.i.d.	13	15	42	36
4	82	16	M	Tacrine (88 weeks)	20 mg t.i.d.	13	10	45	41
5	71	12	F	Tacrine (46 weeks)	20 mg q.i.d.	17	17	41	40
6	70	12	F	Tacrine (78 weeks)	20 mg q.i.d.	23	23	24	27
7	79	14	M	Tacrine (31 weeks)	20 mg q.i.d.	17	19	32	34
8	72	10	F	Tacrine (72 weeks)	20 mg q.i.d.	5	3	53	50
9	54	16	F	Physostigmine (16 weeks)	12 mg b.i.d.	14	16	38	32
10	64	16	F	Physostigmine (26 weeks)	15 mg b.i.d.	23	22	28	29

^aMean=68.6 years (SD=8.5).^bMean=13.7 years (SD=2.0).^cHigher scores indicate better performance.^dLower scores indicate better performance.^eSubjects 1, 2, 4, 5, and 7 received *l*-deprenyl during the first period.

50 years old or older, good physical health verified by examination and by screening laboratory studies, diagnosis of probable Alzheimer's disease according to the criteria of the National Institute of Neurological and Communicative Disorders and Stroke and the Alzheimer's Disease and Related Disorders Association, computerized tomography or magnetic resonance imaging scan consistent with the diagnosis of Alzheimer's disease, and provision of informed consent. Exclusion criteria were insulin-dependent diabetes, hypertension, other CNS disorders, head injury, DSM-III-R axis I disorder other than primary degenerative dementia, clinically significant systemic disease, modified Hachinski ischemic score greater than 4, use of medication with intrinsic CNS activity, and blood pressure greater than 170 mm Hg systolic or 100 mm Hg diastolic.

Procedure

Subjects were assessed before receiving *l*-deprenyl or placebo (baseline) and at the end of each 4-week period by a psychometrician who was blind to treatment assignment. The psychometrician used the cognitive subscale of the Alzheimer's Disease Assessment Scale (7) and the Mini-Mental State examination (4) as outcome instruments. Equivalent versions of the Alzheimer's Disease Assessment Scale were used at each testing interval.

Analysis

Both analysis of variance (ANOVA) and the non-parametric signed rank test were used to compare each subject's performance while taking *l*-deprenyl and while taking placebo. The signed rank test was used on the difference scores because it better controls for type I error, and the ANOVA was used to assess the probability of carryover effects (8). Also, change scores were analyzed only for the comparison of the group that first

received *l*-deprenyl with the group that first received placebo, as previously recommended (8).

RESULTS

The patients included in the study were six women and four men; their mean age was 68.6 years (SD=8.5). Their mean baseline score on the cognitive subscale was 35.2 (SD=8.3), and their mean Mini-Mental State examination score was 15.7 (SD=4.8). Seven subjects were receiving tacrine, and three were receiving physostigmine. Table 1 shows each patient's dose and duration of previous cholinesterase treatment.

The patients' mean improvement on the cognitive subscale was 2.50 points (SD=3.69) ($F=4.11$, $df=1, 8$, $p=0.08$) (Wilcoxon $T+=45.5$, $p=0.04$). There was no significant period effect ($F=0.06$, $df=1, 8$, $p=0.81$) (8). Analysis of the first period, comparing scores on the cognitive subscale in subjects who received *l*-deprenyl first with those who received placebo first, revealed a significant effect for *l*-deprenyl (mean=-1.80, SD=2.86, versus mean=3.00, SD=3.39) ($F=5.85$, $df=1, 8$, $p=0.04$).

The correlation between baseline score on the cognitive subscale and change in score with *l*-deprenyl ($r=0.34$, $df=8$, $p=0.33$) was not significant. Inspection of subtests of the cognitive subscale suggested that recognition memory improved the most (mean=1.20, SD=2.39) but not significantly ($t=1.12$, $df=9$, $p=0.32$).

There was no significant improvement in Mini-Mental State examination scores; in fact, there was a small numerical worsening. The mean change in scores for the group was -0.60 (SD=2.17) ($F=0.80$, $df=1, 8$, $p=0.40$) (Wilcoxon $T+=12.0$, $p>0.50$). There was no significant first-period effect for *l*-deprenyl compared with placebo (mean=-1.40, SD=4.83, versus mean=0.00, SD=1.58) ($F=0.38$, $df=1, 8$, $p=0.55$).

There were no side effects other than ongoing, inter-

mittent mild nausea in two physostigmine-treated subjects before, during, and after the trial; one patient had nausea during *l*-deprenyl administration.

DISCUSSION

Although the size of this pilot crossover study precludes generalizations, some comments can be made. At best, the results of crossover studies can only suggest hypotheses for more definitive studies because the possibilities of period effects, carryover effects, and illness progression cannot be excluded as affecting the results (8). Sample selection bias is important to consider because the subjects selected to receive *l*-deprenyl were largely those believed to have benefited from ongoing anticholinesterase treatment. (Treatment had been discontinued in subjects who seemed not to have benefited; these subjects were thus not available for this protocol.) Nevertheless, the magnitude of improvement in cognitive subscale score observed in this study—2.50 points—is similar to that reported with tacrine alone in recent multicenter trials (4, 5). The statistical significance of these results was supported by the statistically significant first-period effect, essentially a 4-week, parallel-group trial.

Because patients had been receiving the cholinesterase inhibitor for 16–88 weeks before entering this study, it cannot be determined whether they were still receiving maximal benefit from it. During their cholinesterase inhibitor trials, however, the scores on the cognitive subscale of eight patients improved and two worsened. It is noteworthy that all but one subject would have still qualified for the previous multicenter trials. This group showed a mean change in score on the cognitive subscale of 4.9 points over a mean of 47 weeks before this study—somewhat less than the expected 7-point yearly decline of untreated patients (4). Therefore, it is conceivable that patients were still deriving benefit from the cholinesterase inhibitor during this study.

Although one interpretation for the improvement associated with *l*-deprenyl is an additive therapeutic effect to that of the cholinesterase inhibitor, it is possible that the *l*-deprenyl alone provided the efficacy. Patients who responded positively to cholinesterase inhibitors also tended to respond to *l*-deprenyl.

The insensitivity to change on the Mini-Mental State examination was not unexpected because treatment effects on the Mini-Mental State examination have been seen generally only in larger trials (4, 5).

Our current results are consistent with the significant findings in previous placebo-controlled trials of *l*-deprenyl alone in patients with Alzheimer's disease (1). They are also consistent with a finding of a trend for improvement in recall on a picture recognition task ($p=0.10$) in a previous placebo-controlled crossover study of *l*-deprenyl combined with physostigmine in 10 patients with Alzheimer's disease (9).

As Sunderland et al. (9) suggested, there is a need for placebo-controlled, parallel-group trials of combination *l*-deprenyl/cholinesterase inhibitor therapy, perhaps comparing the combination with *l*-deprenyl alone and with a cholinesterase inhibitor alone.

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HIV-Related Risk Behaviors Among Psychiatrically Hospitalized Adolescents and School-Based Adolescents

Ralph J. DiClemente, Ph.D., and Lynn E. Ponton, M.D.

The authors compared the responses of 76 adolescents on an inpatient psychiatric service with those of 802 school-based adolescents in the same city regarding HIV risk behaviors. The psychiatrically hospitalized adolescents reported a significantly higher rate of sexual and drug-related behaviors that involve a risk for contracting sexually transmitted disease, including HIV-related illnesses.

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Adolescents have recently emerged as a risk group for HIV infection. Epidemiologic surveillance data have identified a high rate of drug use and sexual risk-taking behavior among this age group (1, 2). Information about the prevalence of adolescents' HIV-related risk taking has been derived primarily from school-based surveys through the Centers for Disease Control (CDC) nationwide surveillance of adolescents' knowledge, attitudes, and HIV-related behaviors (3). The prevalence of HIV-related risk taking, however, is not uniform across adolescent subgroups (4). Less information is available about adolescents who might be at greater risk of HIV infection. One understudied group of adolescents who may be at greater risk of HIV infection are those with severe emotional disturbances requiring inpatient psychiatric hospitalization.

Data are not yet available on the prevalence of HIV-related risk behaviors in psychiatrically hospitalized adolescents. Consequently, it is not possible to assess any differences in risk taking between psychiatrically disturbed adolescents and the general population of adolescents. Such information could be useful in evaluating the risk of HIV infection among psychiatrically hospitalized adolescents as well as in the planning of inpatient HIV prevention programs.

This report determined the rate of HIV-related risk-taking behavior among psychiatrically hospitalized adolescents admitted to a university hospital adolescent psychiatric unit in an AIDS epicenter and compared it with the rate reported in a similar-age school-based group of adolescents.

METHOD

All newly admitted adolescents to an inpatient adolescent psychiatry service between Jan. 1, 1988, and April 30, 1989, were asked to participate in a study to assess HIV-related risk behavior. The project was explained to all of the patients and their parents or guardians, and written consent was required to participate. Seventy-six adolescent inpatients were interviewed. These adolescents represent all consecutive admissions during the specified time period.

The adolescents' HIV-related risk behaviors were assessed by using a structured interview conducted in private as part of the standard intake procedure. The interview assessed a variety of HIV risk behaviors, including age at sexual debut, condom use during vaginal and anal intercourse, number of sex partners, frequency of sexual intercourse, homosexuality, injection drug use, sharing of injection drug needles, and a history of pregnancy (for girls) and sexually transmitted diseases.

To assess differences in HIV-related risk behaviors, the responses to questions about specific behaviors identified among the psychiatrically hospitalized group were compared with those of a school-based group of 802 adolescents in the same city. The school group had participated in the CDC survey monitoring adolescents' knowledge, attitudes, and risk behaviors (3). Participation in this survey required completing an anonymous self-report questionnaire regarding similar sexual and drug-related risk behaviors.

RESULTS

The psychiatrically hospitalized adolescents reported a high rate of sexual and drug risk behaviors associated with HIV acquisition and transmission. Forty (53%) reported being sexually active. The mean age at sexual debut was 11.4; 20 (50%) of the sexually active adolescents had their sexual debut at age 12 or younger. Of

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TABLE 1. Relative Risk of HIV-Related Behaviors Among Psychiatrically Hospitalized Adolescents and a School-Based Comparison Group

Risk Behavior	Psychiatrically Hospitalized Adolescents (N=76)		School Group (N=802)		Prevalence Ratio ^a	95% Confidence Interval
	N	%	N	%		
Sexually active	40	52.6	195	28.6	1.8	1.4–2.3
Use injection drugs	7	9.2	29	3.7	2.5	1.1–5.5
Share injection needles ^b	7	100.0	19	65.5	1.4	1.0–1.9
Have multiple sex partners ^c	25	62.5	101	51.8	1.2	0.9–1.6
Never use condoms ^d	18	45.0	29	24.8	1.9	1.2–3.0

^aCalculated with the school group as the referent for comparison.

^bPercents based on number of respondents who reported injection drug use.

^cMore than three lifetime sex partners; percents based on number of respondents who reported they were sexually active.

^dPercents based on number of respondents who reported they were sexually active.

those who reported being sexually active, only 15 (38%) were monogamous at the time of the interview and only nine (23%) used condoms consistently during vaginal sexual intercourse; 31 (78%) reported rarely or never using condoms during anal sex. Eight (20%) reported past homosexual relationships, and eight (20%) reported having had sex with an injection drug user.

Injection drug use was also prevalent. Seven (9%) reported having used injection drugs, and all seven of these patients reported sharing needles. There was also a strong association between the use of noninjection drugs and high-risk sex: three (15%) of the 20 patients who used noninjection drugs admitted to trading sex for drugs.

Other markers, such as a high lifetime prevalence of sexually transmitted diseases (15%, N=6) and pregnancy among girls (27%, N=6), suggest that the risk of exposure to HIV among psychiatrically hospitalized adolescents is substantial.

The rates of HIV risk behaviors among these psychiatrically hospitalized adolescents were compared with those among a similar-aged school-based group of adolescents. Although the school survey did not include all of the items included in the structured interview given to the inpatients, those items which were common to both the survey and the interview were selected for comparison. To quantify the difference in rate of risk behaviors between psychiatrically hospitalized and school groups, we calculated prevalence ratios and their corresponding confidence limits for each risk behavior for which data were available from both groups.

Psychiatrically hospitalized adolescents had a markedly higher rate of risk-taking behaviors than their school-based peers (table 1). Of special interest is the high rate of injection drug use among the psychiatrically hospitalized group compared with the school group (table 1). The hospitalized adolescents were also 1.8 (almost twice) as likely to be sexually active and almost twice as likely to report not using condoms during sexual intercourse.

CONCLUSIONS

Psychiatrically hospitalized adolescents reported a high rate of HIV-related risk behaviors. Furthermore, their rate of sexual and drug-related risk behaviors was significantly

higher than that of a similar-age referent adolescent group. These findings suggest that adolescents with severe emotional disorders are at considerable risk of HIV infection and that they are potential sources for HIV transmission to other adolescent and adult populations.

These findings suggest that HIV prevention education programs are needed on adolescent inpatient units. These programs should provide adolescents with information about risk behaviors and, more importantly, make adolescents aware of protective measures, such as consistent condom use during sexual intercourse and cleaning injection drug needles with bleach. As important as providing prevention information, programs for psychiatrically hospitalized adolescents must provide skills training in sexual communication and risk-reduction strategies that will promote the use of self-protective behaviors (5, 6). However, these programs, to be effective, must be tailored to the particular needs and developmental level of psychiatrically disturbed adolescents (7).

While the number of adolescents receiving psychiatric treatment at inpatient facilities is relatively small, given the present findings, the potential health threat posed by psychiatrically hospitalized adolescents to themselves and society may far exceed their proportion in the population.

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Manic Syndrome Early and Late in the Course of HIV

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In a chart review at a hospital's infectious disease AIDS clinic, manic syndromes affected 8% of patients who had AIDS. Of the 14 patients with manic episodes, those without a family or personal history of mood disorder presented later in the course of HIV infection and had a higher prevalence of comorbid dementia.
(Am J Psychiatry 1993; 150:326-327)

In 23 case reports of manic syndromes afflicting patients infected with HIV, a strong association was noted between mania and the development of cognitive decline or coarse brain injury (1-9). In contrast, Buhrich et al. (10) presented three cases in which patients developed mania "indistinguishable from functional psychosis." Similar cases have been reported by others (11-14). Little is known about the prevalence, risk factors, and pathogenesis of manic syndromes and bipolar disorder in HIV-infected patients. One recent study suggests that a greater than expected number of manic episodes is seen in these patients (6). This paper discusses our experience at a university hospital with HIV-infected patients who developed manic syndromes.

METHOD

We reviewed the charts of 162 patients referred by primary medical caregivers for psychiatric evaluation by a hospital's AIDS psychiatry service over a 17-month period. We evaluated the patients according to the hospital's standard assessment procedures. Diagnoses were made according to DSM-III-R criteria, and 14 patients who met the criteria for a manic episode were identified. These diagnoses were confirmed by detailed chart review, which showed that the presence of typical signs and symptoms had been recorded in the chart for at least 1 week. Patients with a history of bipolar disorder who were referred for other chief complaints were not included.

RESULTS

Twelve patients suffered from manic syndromes and two from hypomanic syndromes. Both cases of hypomania occurred after treatment with antidepressants and required lithium to induce remission despite the discontinuation of the antidepressants. Four patients had a family history of major depression or bipolar disorder, all in a parent or a sibling. Three patients had personal histories of major depression before the diagnosis of HIV infection. Two had personal histories of bipolar disorder. Because there was overlap, a total of seven patients had a family or personal history of mood disorder. No patients suffered from a concurrent substance use or axis II disorder.

The 14 patients were divided into two groups on the basis of the presence (N=7) or absence (N=7) of a personal or family history of affective disorder (see table 1). The groups were similar in terms of demographic characteristics and risk factors for HIV infection. In contrast, fewer patients with a family or personal history of mood disorder had AIDS ($p=0.01$, Fisher's exact test) and a CD4+ cell count below 100 μl ($p=0.06$, Fisher's exact test). CD4+ counts for three patients and brain imaging studies of five patients were not available and were not included in these comparisons.

All but one of the patients in the group without a personal or family history of mood disorder received the clinical diagnosis of dementia ($p=0.004$, Fisher's exact test). The etiology of dementia was uncertain. Of these patients, three had detailed neuropsychological testing that confirmed the diagnosis of dementia. Brain CT scans in two patients and magnetic resonance imaging (MRI) in one patient showed diffuse subcortical white matter atrophy. Results of brain CT scans in two others and brain MRI in one other were normal. The patient who did not suffer from dementia was suspected of having this diagnosis and had a left basal ganglia hypodensity on CT scan. One patient in the group with a personal or family history of mood disorder developed dementia and a right frontal lesion on MRI a year

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TABLE 1. Demographic and Clinical Characteristics of 14 Patients With Mania Who Were Seen at an AIDS Clinic

Item	Number of Patients ^a	
	No History of Mood Disorder (N=7)	History of Mood Disorder (N=7)
Male	7	5
HIV risk factor		
Homosexual	4	3
Intravenous drug use	2	4
HIV stage at diagnosis of mania		
Asymptomatic	0	5
AIDS	7	2 ^b
CD4+ count at diagnosis of mania		
Less than 100 μ l	6	2 ^c
More than 100 μ l	0	3
Dementia at diagnosis of mania	6 ^d	0

^aFor patients with no history of mood disorder, mean age=33.6 years, SD=5.4; for patients with a history of mood disorder, mean=35.1 years, SD=9.0.

^bp=0.01; all analyses by Fisher's exact test.

^cp=0.06.

^dp=0.004.

after diagnosis. Two patients in this group had normal brain CT scans. The latter difference was not significant by Fisher's exact test; however, data were missing on five patients, and the lack of significance may be due to the small size of the groups (type II error).

COMMENT

The hospital's infectious disease AIDS clinic evaluated 1,000 patients during the study period; the 14 referred patients with manic syndrome resulted in a 17-month prevalence of 1.4%. The majority of patients were at the asymptomatic seropositive stage, whereas most referred patients, including those with manic syndromes, suffered from AIDS. In patients who suffered from AIDS, we found nine cases of manic syndrome in a clinic that treated 112 patients with AIDS during the same period, a rate of 8%. This is more than 10 times the 6-month prevalence for manic episode reported in the general population (15).

As HIV affects subcortical brain areas important to the regulation of mood and affect, this may lead to dysregulation of affect, followed by mania or depression, in addition to cognitive decline. This could account for manic syndromes in the group without a personal or family history of mood disorder and the similar cases reported in the literature. Prospective study, with careful psychiatric and neuropsychological assessment, and brain imaging are necessary to confirm this impression.

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Treatment of Alcoholism Among Schizophrenic Outpatients: 4-Year Outcomes

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In this pilot study, the authors assessed 4-year outcomes for 18 schizophrenic outpatients with alcoholism who were treated in an innovative dual-diagnosis program. Over half (61.1%) achieved stable remissions from alcoholism. The mean duration of remission was 26.5 months. (Am J Psychiatry 1993; 150:328-329)

The high rates of comorbidity and adverse consequences of alcoholism in schizophrenic patients are now widely recognized (1). Because these dually diagnosed patients have poor short-term outcomes in traditional mental health programs and do not readily fit into traditional substance abuse treatment programs, the general view of treatment is pessimistic (2). Limited research suggests, however, that integrating substance abuse treatments into mental health programs may be more effective. Kofoed et al. (3) and Hellerstein and Meehan (4) found that dually diagnosed outpatients who participated in substance abuse treatment groups had lower rates of rehospitalization after group treatment began, and Ries and Ellingson (5) found that attending inpatient drug and alcohol discussion groups was associated with short-term abstinence after discharge.

Among state mental health systems, New Hampshire's has taken the lead in integrating substance abuse treatments into mental health programs. In the New Hampshire model (6) interdisciplinary teams of clinicians deliver both mental health and substance abuse treatments in the community. The substance abuse treatment, delivered in individual and group sessions, is specifically tailored for people with severe and persistent mental illness. The purpose of this pilot study was to assess the 4-year outcomes of treatment for alcoholism in schizophrenic patients in the New Hampshire program.

METHOD

The subjects of this report were 18 patients who met the DSM-III-R criteria for both schizophrenia and current alcohol use disorder. They were selected on the basis of a research evaluation of all outpatients with clinical diagnoses of schizophrenia in a rural community mental health center (CMHC) in 1987 (7). One of the 19 patients originally diagnosed with both schizophrenia and active alcohol use disorder died of causes unrelated to substance abuse in 1988. The remaining 18 patients were treated continuously between 1987 and 1991 in an integrated dual-diagnosis program.

These 18 patients had a mean age of 37.9 years ($SD=12.2$) at the time of the 1987 (baseline) evaluation. There were 12 men (66.7%) and six women (33.3%). All were Caucasian. Only two patients (11.1%) were currently married in 1987, 12 (66.7%) had never been married, and four (22.2%) were separated, divorced, or widowed. Their DSM-III-R diagnoses were as follows: schizophrenia, 83.3% ($N=15$); schizoaffective disorder, 16.7% ($N=3$); alcohol use disorder, 100% ($N=18$); and marijuana use disorder, 22.2% ($N=4$).

The 18 subjects were evaluated continuously by clinicians and were formally reevaluated with similar methods and measures by our research team 4 years after their original assessments. Alcohol and drug use were assessed through a combination of psychiatric interviews, clinicians' ratings, clinical records, and intensive case reviews. Remission was defined as abstinence or the absence of alcohol abuse for at least 6 months, as specified in DSM-III-R. One of us (D.L.N.) conducted psychiatric interviews by using the alcohol and drug section of the Structured Clinical Interview for DSM-III-R—Patient Version (8). Case managers rated alcohol and other drug use, on 5-point clinician rating scales (7), on the basis of clinical interviews, longitudinal observations in the community, and information from housing staff, family members, and other people familiar with the patient. All inpatient and outpatient

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records were reviewed to verify remissions. As in the baseline assessment (7), disagreements among these perspectives were resolved by intensive case review. Only one disagreement occurred, and it was easily resolved.

RESULTS

Of the 18 subjects, 11 (61.1%) showed no evidence of alcohol abuse for at least 6 months and were therefore classified as in remission. A 12th subject had used alcohol only once during the previous 6 months but had become intoxicated and verbally abusive toward his family during this episode and was therefore classified as not in remission. The mean length of remission was 26.5 months ($SD=13.5$). Three subjects who failed to attain remission also continued to abuse marijuana.

Since the 18 patients in the study group were engaged in treatment throughout the 4-year interval, remission always occurred in the context of active treatment. All patients received assertive case management (at least weekly visits with a case manager), antipsychotic medications, housing supports, and other CMHC services. In addition, all 18 received behaviorally oriented substance abuse counseling in individual sessions, and 13 patients (72.2%) regularly attended dual-diagnosis groups in the CMHC. Despite encouragement from clinicians, only one patient (5.6%) regularly attended self-help groups such as Alcoholics Anonymous, and he did not attain remission. Several patients were referred to inpatient substance abuse programs, but only one patient (5.6%) completed such a program, and he also failed to attain remission.

DISCUSSION

To our knowledge, this is the first report of the long-term treatment of alcohol-abusing schizophrenic patients. The high rate of remission from alcoholism in this group contrasts markedly with the poor short-term outcomes among dually diagnosed patients in other studies and compares favorably with the rates of stable remission found in several long-term studies of alcoholism treatment (9).

We hypothesize that the substance abuse treatment in our dual-diagnosis program stimulated remission by offering a consistent, longitudinal approach to recovery. One critical outcome of the program was that assertive

outreach ensured that every patient, even those who were initially denying problems and exhibiting non-compliance, became engaged over time in substance abuse treatment within the CMHC. On the other hand, attempts to link patients with the traditional substance abuse treatment system, including inpatient 12-step programs and self-help groups, were unsuccessful.

The results reported here were obtained in an open trial using one clinical site, a small number of subjects, and nonblind raters. In addition to dual-diagnosis treatment, the patients in this pilot study had the advantages of rural living, access to housing, and minimal exposure to more dangerous substances of abuse, such as crack cocaine, that are common in large urban areas (10). These results should be replicated in larger subject groups with controlled trials and analyses of costs. Nonetheless, these outcomes are cause for optimism since the patients were not selected for treatment readiness, were denying alcohol-related problems at baseline (7), and were therefore similar to other schizophrenic patients with alcoholism in the community. Moreover, the long-term perspective used in this evaluation indicates that remission can be stable and long-lasting.

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Correlation of Severity of Psychiatric Patients' Delusions With Right Hemispatial Inattention (Left-Turning Behavior)

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Studies associate psychotic disorders with various forms of subtle inattention to the right hemispace (left-turning behavior). The authors examined the correlation between this dopamine-related sign and severity of delusions (presumably dopaminergic symptoms) in 20 psychotic patients. Delusions were significantly correlated with severity of left-turning bias, and this neurological sign accounted for 33% of the variance in severity of delusions.
(Am J Psychiatry 1993; 150:330-332)

Over the last 5 years, several studies of unmedicated schizophrenic patients have identified various forms of subtle inattention to the right hemispace (i.e., a spontaneous, subtle preference for turning toward the left hemispace while moving about) (1-3). Hemispatial attention and inattention (turning asymmetry) are partly controlled by ascending subcortical dopaminergic systems (4, 5). Since delusions are among the few symptoms of schizophrenia that are responsive to dopamine blockers, we hypothesized that left-turning asymmetry would correlate with severity of delusional symptoms.

METHOD

The subjects of this study were 20 research inpatients (18 male and two female), ranging in age from 15 to 70 years, who were assessed by a neuropsychiatric special evaluation unit of a VA medical center. Each had received a diagnosis of schizophrenia on the basis of the Structured Clinical Interview for DSM-III-R—Patient Version (SCID-P) (6), with the exception of one patient who was diagnosed as having alcohol hallucinosis and significant delusions. All patients except one had been free of neuroleptic medications for at least 1 month before entering the study. The exception was a patient hospitalized for the first time who had never previously taken antipsychotic medication and who was in her third day

of taking a neuroleptic when she started the study. Each patient had a complete psychiatric and neurological examination, laboratory tests, urinalysis, and urine toxicology screening. The patients were administered the Scale for the Assessment of Positive Symptoms (7), the Scale for the Assessment of Negative Symptoms (8), and the Brief Psychiatric Rating Scale (BPRS) (9) on the first day of the study. Both right-handed subjects (N=17) and left-handed subjects (N=3) were included because several studies have shown that subjects' handedness has surprisingly little effect on turning behavior (1, 10, 11).

The mean age of the subjects was 37 years (SD=11), the mean number of months since they had last taken neuroleptics was 26 (SD=37), and the mean number of years they had been ill was 13 (SD=9). The subjects' mean scores on the BPRS and the scales for negative and positive symptoms were, respectively, 41 (SD=12), 35 (SD=18), and 44 (SD=18).

After giving informed consent, the subjects were asked to wear a monitoring device on their belts during waking hours. The device has been described previously (1, 10, 11). The activity monitors were worn for a mean of 93 hours (SD=46), and the mean total number of turns recorded was 872 (SD=619). A minimum of 200 full turns was required for a subject to be included in the study. The requirement was met by patients wearing the monitor for 2-8 days. The subjects were asked to go about their usual activities and were unaware of the nature of the device.

We chose a priori to use the Scale for the Assessment of Positive Symptoms for measuring delusional symptoms because of the great detail obtainable with the use of this scale, which includes 12 questions on such symptoms. We computed a mean score for the 12 questions, henceforth denoted as the mean delusion score. The mean delusion score for the group on the Scale for the Assessment of Positive Symptoms was 1.4 (SD=0.8).

The index of turning (circling) behavior was percent

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right turns (1), defined as the number of 360° turns toward the right hemisphere, divided by the total number of full 360° turns in either direction, multiplied by 100. Therefore, percent right turns reflects hemispatial-preference asymmetry and is independent of total activity. Values between 0% and 40% reflect varying degrees of right hemispatial inattention (right hemispatial neglect), and values between 60% and 100% reflect varying degrees of left hemispatial inattention (left hemispatial neglect) (1, 10, 11).

We hypothesized that in unmedicated psychotic patients there would be an inverse relation between right-turning preference (percent right turns) and the severity of delusions as recorded by the mean delusion score on the Scale for the Assessment of Positive Symptoms. We hypothesized that the severity of left-turning behavior is indicative of the severity of delusional symptoms and that subjects with lower percent right turns (i.e., marked preferential turning to the left) would manifest higher delusion scores. To test this hypothesis, we calculated a regression equation, using percent right turns as a predictor of the mean delusion score. Two additional regression analyses were obtained with the use of the more general indexes of psychosis (total score on the Scale for the Assessment of Positive Symptoms) and of psychopathology (total BPRS score).

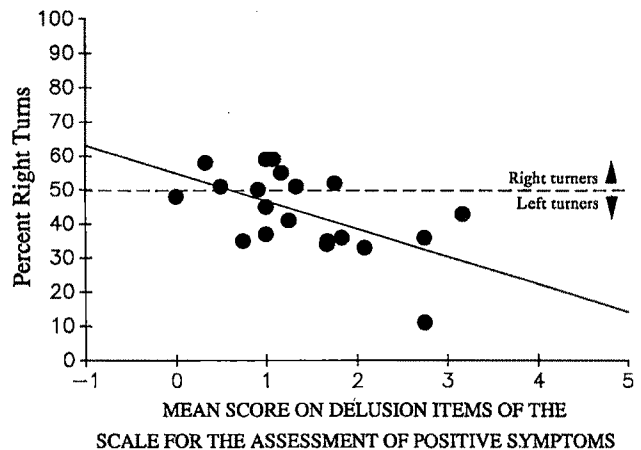
One subject exhibited extreme left-turning behavior (percent right turns=11, almost 3 standard deviations from the mean). Because of the small size of the study group, we recalculated the analysis after removing the data on this subject. Because the hypothesis was directional (i.e., that there would be an inverse relation between percent right turns and mean delusion score), all analyses were calculated with a one-tailed probability value.

RESULTS

The linear regression, with the mean delusion score the dependent variable and percent right turns as the predictor, was significant ($F=8.67$, $df=1, 18$, $p=0.004$; $r=-0.57$). The results remained significant even after the removal of the one subject exhibiting extreme left-turning asymmetry ($F=4.46$, $df=1, 17$, $p<0.03$). Figure 1 presents a scatterplot of the data and the regression line of best fit. Turning behavior accounted for 33% of the variance in mean delusion scores (29% when the adjusted value was used).

Scores on the Scale for the Assessment of Positive Symptoms and especially scores on the BPRS are less affected by delusional symptoms alone (e.g., only one of the 18 items on the BPRS addresses delusions). As expected, the regression analysis with turning behavior as a predictor of total score on the Scale for the Assessment of Positive Symptoms was in the expected direction and was nonsignificant ($F=0.85$, $df=1, 18$, $p=0.18$; $r=-0.21$). Also as expected, turning behavior did not predict total BPRS score ($F=0.07$, $df=1, 18$, $p=0.39$; $r=0.06$).

FIGURE 1. Left Turning as a Predictor of Delusional Ideation in 20 Neuroleptic-Free or Neuroleptic-Naive Psychotic Subjects



DISCUSSION

This study demonstrated a statistically significant relation between severity of delusions and left-turning behavior (right hemispatial inattention) in a group of 20 psychotic patients. Our results are consistent with reports of left-turning behavior (1) and other subtle forms of right hemispatial inattention (2, 3) in unmedicated and medication-naïve patients with schizophrenia. The present study, however, had a different purpose. By design, it included both severely delusional and nondelusional schizophrenic patients. This wide range of severity of delusions permitted us to demonstrate the correlation of the two (presumably dopamine-related) phenomena.

Hemispatial inattention and delusional symptoms often appear together in neurological patients (12–14). As has been pointed out elsewhere, two neurological conditions that can produce psychotic states in humans (right frontoparietal pathology and left amygdala kindling) have been shown to result also in left-turning behavior (13, 15). Studies also associate such persistent attentional asymmetry with severe bilateral cortical pathology or cortical immaturity (5, 12, 14), and some have speculated that hemispatial inattention is a key concept for understanding the neurology of psychosis (13, 15). Being a sign, rather than a symptom, hemispatial inattention may often be easier to document than delusions in a psychotic patient. Ongoing studies are examining the potential value of hemispatial inattention in predicting response to dopamine blockers and outcome in psychosis.

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Correlation Between Antisaccade and Wisconsin Card Sorting Test Performance in Schizophrenia

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In 27 patients with chronic schizophrenia, there was a significant correlation between performance on an antisaccade eye movement task and on the Wisconsin Card Sorting Test. A significant correlation was not obtained between antisaccade task performance and scores on the modified Mini-Mental State examination or the Schedule for the Assessment of Negative Symptoms in Schizophrenia. In addition, patients' antisaccade task performance was impaired compared with that of 12 normal subjects.

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Recent work has reported a significant correlation between smooth pursuit eye movement and Wisconsin Card Sorting Test performance in patients with schizophrenia (1). Performance on both the smooth pursuit eye movement and Wisconsin Card Sorting Test tasks is thought to depend on intact frontal lobe functioning (1). Impaired performance on another eye movement task, namely, the antisaccade task, has also been shown to be related to frontal lobe dysfunction (2). In the antisaccade task, subjects need to suppress reflexive eye movements toward a cue that they have been instructed not to look at, but rather to look in the opposite direction of the cue. Schizophrenic patients have more difficulty suppressing reflexive glances at the cue than do nonschizophrenic control subjects (3); 73% of the patients with impaired antisaccade task performance showed atrophy of the frontal cortex on computed tomography scans.

In this study, we examined the relationship between antisaccade task performance in patients with schizophrenia and other components of the schizophrenic syndrome that are thought to reflect impaired frontal lobe function, specifically, impaired Wisconsin Card Sorting Test performance and the so-called negative symptoms of schizophrenia, such as alogia, lack of motivation, and affective flattening (4). We also compared performance on the antisaccade task between schizophrenic and normal subjects.

METHOD

Twenty-seven patients (26 men, one woman; age range=26-59 years) who fulfilled DSM-III-R criteria for chronic schizophrenia and who gave written informed consent to participate were selected for the study. In all cases, a consensus diagnosis was made by at least two psychiatrists after the patient interview and chart review. The mean age of the patients was 38.5 years (SD=7.3), and the mean duration of illness was 14.8 years (SD=5.8). At the time of testing, patients were being treated with conventional neuroleptic medications (mean dose=1394.0 mg in chlorpromazine equivalents, SD=1560.2). Patients were clinically stable and fully able to cooperate with testing. The clinical diagnostic interview also included the Brief Psychiatric Rating Scale (BPRS) (5), the Schedule for the Assessment of Negative Symptoms in Schizophrenia (6), and the modified Mini-Mental State examination (with a total of 35 possible points) (7). The scores for the BPRS and Schedule for the Assessment of Negative Symptoms in Schizophrenia were determined by the consensus of at least two psychiatrists who were blind to the patients' antisaccade and Wisconsin Card Sorting Test performance. Patients provided a medical history and received a physical examination and laboratory screen. They were free of medical or neurological conditions that could contribute to central nervous system or oculomotor impairment such as AIDS, stroke, seizure disorder, or hypoglycemia. Patients with substance use dependence disorders within the past year were excluded. Twelve normal subjects were tested on the antisaccade paradigm and Wisconsin Card Sorting Test. These comparison subjects included unpaid volunteers from the hospital staff, as well as paid volunteers who answered an advertisement. Comparison subjects did not have a past history of psychiatric illness or a past or

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present history of drug or alcohol abuse and had not used alcohol in the 24 hours before testing. All subjects were required to be able to clearly visualize and identify stimuli on the computer screen. Subjects who did not have a full range of extraocular eye movements or who had nystagmus or diplopia were excluded. The age of the comparison subjects (mean=37.6 years, SD=12.0) did not differ significantly from the age of the schizophrenic patients ($t=0.30$, $df=37$, $p=0.77$). There was a marginally significant difference between patients (mean=12.6 years, SD=2.0) and normal subjects (mean=14.4 years, SD=3.5) in number of years of education completed ($t=1.96$, $df=35$, $p=0.06$). For two patients, data on level of education were not available.

Antisaccade performance was evaluated with an infrared scleral reflectance system. The stimuli for this task, based on an antisaccade task previously described (2), were presented on a computer screen positioned 100 cm from the subject. The series of events in one trial of the antisaccade task (and their duration) were as follows: 1) cross-shaped center fixation point (1.5 seconds), 2) blank screen (1.5 seconds), 3) open "cue" box 7.5° to the left or right of the center (1.0 second), 4) blank screen (0.5 second), 5) "target" box filled with an X or O symbol appearing on the opposite side from the cue box equidistant from the center (1.0 second), and 6) blank screen (1.5 seconds). The next trial began again with the center fixation point. The open box was about 2° by 2° in size; the X and O symbols were about 1.5° in size (2). Across all trials, there were 20 presentations of the X symbol and 30 of the O symbol. Subjects were instructed *not* to look at the cue (open box), but rather to look immediately in the opposite direction, at approximately an equal distance from where the center cross appeared. They were told that a box filled with an X or O symbol would appear on the screen on the opposite side from the cue and that they should press a button on the response box when the symbol was an X. Due to a technical failure in recording, data on symbol identification were not available for the first 10 schizophrenic patients tested. Formal measurement of the antisaccade task was not begun until subjects completed an antisaccade stimulus demonstration and it was clear that they understood the task. There were 50 trials in the task: 25 cue presentations to the left side of the screen and 25 to the right, presented in a random order. In the antisaccade task, the number of eye movements toward the cue (i.e., reflexive glances) was scored. Each subject was also tested with the Wisconsin Card Sorting Test, using a standardized administration and scoring system (8). The modified Mini-Mental State examination, BPRS, and Schedule for the Assessment of Negative Symptoms in Schizophrenia were not administered to comparison subjects and to one schizophrenic subject.

RESULTS

The number of reflexive eye movements toward the cue during the antisaccade task was significantly higher for schizophrenic patients (mean=38.9, SD=8.9) than

for comparison subjects (mean=29.3, SD=13.0) ($t=-2.7$, $df=37$, $p=0.01$). There was no significant difference in the X symbol identification between the two groups (total possible correct=20; schizophrenic patient group mean=19.5, SD=1.1; normal group mean=19.9, SD=0.3; $t=1.22$, $df=27$, $p=0.23$). The mean number of perseverative errors on the Wisconsin Card Sorting Test for the patient group was 43.1 (SD=28.8), and the mean number of categories completed was 2.3 (SD=2.4); both of these measures were significantly correlated with the number of reflexive glances during the antisaccade task ($r=0.57$, $df=25$, $p<0.01$ and $r=-0.51$, $df=25$, $p<0.01$, respectively). For the comparison group, the mean number of perseverative errors on the Wisconsin Card Sorting Test was 19.2 (SD=11.7), and the mean number of categories sorted was 4.6 (SD=2.0). Neither of these measures correlated with the number of reflexive glances during the antisaccade task in this group (perseverative errors: $r=-0.13$, $df=10$; categories: $r=0.04$, $df=10$).

In the patients with schizophrenia, the mean BPRS score was 49.6 (SD=12.0), the mean total score on the Schedule for the Assessment of Negative Symptoms in Schizophrenia was 46.8 (SD=19.1), and the mean score on the modified Mini-Mental State examination was 31.3 (SD=3.5). A significant correlation was not found between antisaccade performance and scores on the BPRS ($r=-0.19$, $df=24$), Schedule for the Assessment of Negative Symptoms in Schizophrenia ($r=0.24$, $df=24$), and modified Mini-Mental State examination ($r=-0.17$, $df=24$). In addition, neuroleptic dose was not significantly correlated with performance on the antisaccade task ($r=0.29$, $df=25$), Wisconsin Card Sorting Test (perseverative errors: $r=0.12$, $df=25$; number of categories sorted: $r=-0.01$, $df=25$), or modified Mini-Mental State examination ($r=-0.01$, $df=24$). Antisaccade performance was also not significantly correlated with level of education in either the schizophrenic group ($r=0.20$, $df=23$) or comparison group ($r=0.20$, $df=10$). Finally, a significant correlation between scores on the Schedule for the Assessment of Negative Symptoms in Schizophrenia and performance on the Wisconsin Card Sorting Test was not observed in the schizophrenic group (perseverative errors: $r=0.02$, $df=24$; categories: $r=-0.23$, $df=24$).

DISCUSSION

The results of this study show that schizophrenic patients were impaired on an antisaccade task compared with normal subjects, as has been reported previously (3). In this study, we report a correlation in schizophrenic patients between performance on an antisaccade task and the Wisconsin Card Sorting Test. Because damage in or about the dorsolateral prefrontal cortex has been implicated in deficits on both of these tasks (2, 9), our findings might point to a relationship between eye movement deficits and frontal lobe dysfunction in schizophrenia. Moreover, we found that antisaccade performance was not significantly correlated with

BPRS or modified Mini-Mental State examination scores, education level, or neuroleptic dose. These results suggest that impairment on the antisaccade task is not simply related to the patients' overall level of psychopathology, intellectual or cognitive ability, or dose of medication. Furthermore, performance on the Wisconsin Card Sorting Test and antisaccade task was unrelated in the normal subjects. It is of interest that Litman et al. (1) also reported no correlation between smooth pursuit eye movement and Wisconsin Card Sorting Test performance in their normal comparison subjects. However, the results of these studies should be interpreted with caution because of the small group sizes and the possibility of type II error.

The magnitude of the correlation found in the present study is similar to one reported in an earlier study (1) between smooth pursuit eye movement and Wisconsin Card Sorting Test performance. Specifically, Litman et al. (1) found an inverse correlation of -0.60 between better smooth pursuit eye movement gain and perseverative errors on the Wisconsin Card Sorting Test and a correlation of 0.66 between smooth pursuit eye movement gain and the number of categories sorted on the Wisconsin Card Sorting Test. We observed a correlation of comparable magnitude between poorer antisaccade performance (as measured by the number of reflexive glances) and perseverative errors ($r=0.57$). We also found a significant inverse correlation ($p<0.01$) between poorer antisaccade performance and the number of categories sorted on the Wisconsin Card Sorting Test, but the magnitude was somewhat lower than that reported by Litman et al. (1) ($r=-0.51$ versus $r=0.66$). The results of these studies suggest that both the smooth pursuit and antisaccade tasks may reflect neuropsychological deficits related to frontal lobe dysfunction in schizophrenia.

Although it has been hypothesized that negative symptoms reflect frontal lobe dysfunction (4), we did not obtain a significant correlation between severity of negative symptoms and performance on either the antisaccade task or the Wisconsin Card Sorting Test. It should be noted, however, that other investigators have found significant relationships between frontal lobe neuropsychological dysfunction and negative symptoms (9). Further research is needed to clarify the relationship among eye movement deficits, neuropsychological deficits, and symptoms of schizophrenia.

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Blunted Growth Hormone Responses to Growth Hormone-Releasing Factor and to Clonidine in Panic Disorder

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Blunted growth hormone (GH) responses to growth hormone-releasing factor (GH-RF) and clonidine have been reported in patients with panic disorder. In this study GH-RF and clonidine were administered to 13 patients with panic disorder and 20 healthy volunteers. Compared to the normal subjects, the patients with panic disorder had significantly blunted GH responses after both GH-RF and clonidine.
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The growth hormone (GH) response to the α_2 -adrenergic agonist clonidine has been used as an indirect measure of central noradrenergic activity in patients with panic disorder (1-5). Despite the complexity of GH regulation, "blunted" GH response to clonidine has usually been attributed to postsynaptic noradrenergic down-regulation (6). Diminished or blunted GH response to clonidine challenge was noted in four of five reports on patients with panic disorder (1-3, 5). The single study that used growth hormone-releasing factor (GH-RF) in patients with panic disorder reported a blunted GH response (7).

The goal of this study was to replicate the GH response to GH-RF reported by Rapaport et al. (7) and to examine the pattern of GH responses to infusions of both clonidine and GH-RF within the same subject.

METHOD

Thirteen outpatients (nine women and four men) meeting the DSM-III-R criteria for panic disorder (mean age=33 years, SD=9, range=20-44) participated in the study. The patients had no concurrent affective illness and had been medication free for at least 4 weeks before the study. Twenty normal volunteers, seven women and 13 men (mean age=30 years, SD=10, range=20-44), who were free of psychopathology served as the comparison group. Although there were trends for the patients to be older than the comparison group ($t=1.8$, $df=31$, $p=0.08$) and for the proportion of men to be

greater in the normal group ($p=0.08$, Fisher's exact test), these variables did not differ significantly between groups. All subjects were physically healthy and gave both oral and written informed consent. All of the women were premenopausal, and the study was completed during the first 10 days of the menstrual cycle. The GH responses to clonidine and GH-RF in the normal group have been previously reported (8).

All subjects received a low-monoamine diet for at least 72 hours prior to the study. Subjects received 2 $\mu\text{g/kg}$ of clonidine, 1 $\mu\text{g/kg}$ of GH-RF, or placebo following an overnight fast. Infusions were administered in randomized order in double-blind fashion. Blood was drawn at baseline for GH determination and at 15-minute intervals (15, 30, 45, and 60 minutes) following the infusion. GH levels were assayed by a radioimmunoassay technique with interassay and intra-assay variabilities of 7.3% and 4.9%, respectively.

Clonidine infusion data from one patient with panic disorder who had an elevated baseline GH level (>3 ng/ml) were not used (6). Only GH-RF and clonidine infusion data are presented here, as there were no significant effects of placebo. Since GH secretion is not normally distributed, the GH data were logarithmically transformed for the analysis of variance (ANOVA) with repeated measures. Maximal change in GH secretion (Δ_{max} GH) and area under the GH secretory curve (GH_{AUC}) were compared using the Mann-Whitney U test on nontransformed data.

The proportions of patients and comparison subjects with "positive" GH responses (doubling of baseline GH level and an absolute increase in GH level of >5 ng/ml) were compared using Fisher's exact test, two-tailed. Comparisons of the correlation coefficients for the GH_{AUC} response to GH-RF and clonidine in the panic disorder group and the normal group were performed using the z transformation described by Edwards (9).

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RESULTS

The patients with panic disorder, compared to the normal subjects, had significantly blunted GH responses to GH-RF according to the ANOVA and the analyses of Δ_{\max} GH, GH_{AUC} , and the proportions of subjects with an operationally defined positive GH response.

The ANOVA revealed a significant Diagnosis by Time interaction ($F=3.0$, $df=4$, 124 , $p=0.02$). Post hoc analysis revealed that the GH response to GH-RF was significantly lower in the patients with panic disorder than in the normal subjects at 30 minutes ($t=-2.3$, $df=31$, $p<0.03$) and 45 minutes ($t=-2.6$, $df=31$, $p<0.02$) following GH-RF administration.

Mean Δ_{\max} GH following GH-RF was 5.5 ng/ml ($SD=8.0$) in the patients with panic disorder and 8.0 ng/ml ($SD=5.9$) in the comparison subjects (Mann-Whitney $U=180$, $p<0.03$). Mean GH_{AUC} following GH-RF was 188 ng \times min/ml ($SD=301$) in the patients with panic disorder and 294 ng \times min/ml ($SD=271$) in the normal group (Mann-Whitney $U=194$, $p<0.02$).

A positive GH response to GH-RF was observed in three (23%) of the 13 patients with panic disorder, compared to 13 (65%) of the 20 normal subjects ($p=0.03$, Fisher's exact test).

The patients with panic disorder also exhibited blunted GH responses to clonidine compared with the responses of the normal subjects. The ANOVA revealed a significant Diagnosis by Time interaction ($F=7.1$, $df=4$, 120 , $p<0.001$). Post hoc analysis demonstrated a significantly blunted GH response to clonidine in the patients compared with the normal subjects at 30 minutes ($t=-2.0$, $df=30$, $p=0.05$), 45 minutes ($t=-2.6$, $df=30$, $p=0.01$), and 60 minutes ($t=-2.6$, $df=30$, $p<0.02$).

Mean Δ_{\max} GH following clonidine was 2.6 ng/ml ($SD=3.9$) in the patients with panic disorder and 6.5 ng/ml ($SD=6.0$) in the normal subjects (Mann-Whitney $U=177$, $p<0.03$). Mean GH_{AUC} following clonidine was 83 ng \times min/ml ($SD=137$) in the patients with panic disorder and 195 ng \times min/ml ($SD=209$) in the normal group (Mann-Whitney $U=173$, $p<0.04$).

Two (17%) of 12 patients with panic disorder and 12 (60%) of the 20 normal subjects had positive GH responses to clonidine ($p<0.02$, Fisher's exact test).

The GH responses to GH-RF and clonidine did not differ when the magnitude of the GH responses was compared or when the rate of positive responders was compared. One (11%) of 12 patients with panic disorder and 10 (50%) of the 20 normal subjects had positive GH secretory responses to both probes ($p=0.02$, Fisher's exact test).

There was a significant positive correlation between GH_{AUC} following GH-RF infusion and GH_{AUC} following clonidine infusion in the combined groups (panic disorder plus normal comparison subjects) ($r_s=0.57$, $N=32$, $p<0.001$) as well as in the normal comparison group alone ($r_s=0.56$, $N=20$, $p<0.02$). In the panic disorder group, however, the relationship failed to reach statistical significance ($r_s=0.48$, $N=12$, $p<0.10$). The z transformation of the correlation coefficients (panic disorder, $r_s=0.56$; normal sub-

jects, $r_s=0.48$; $z=0.2$, $p=0.80$) indicated that the correlation values did not differ across diagnostic groups (9).

DISCUSSION

Our findings replicate the blunted GH response to GH-RF reported by Rapaport et al. (7) and further support the previous findings of blunted GH response to clonidine in patients with panic disorder (1-3, 5). This is the first study to date to report blunted GH responses to both secretagogues in the same patients with panic disorder. These results should be replicated in a larger study group.

Patients with major depression have been reported to exhibit blunted GH responses to both GH-RF and clonidine (10), while patients with anorexia nervosa have normal GH responses to clonidine and exaggerated GH responses to GH-RF. Patients with panic disorder, therefore, appear to share the neuroendocrine response pattern of patients with major depression (6).

While the findings we have reported are consistent with an abnormal α_2 -adrenergic system in patients with panic disorder, further examination of the hypothalamic-pituitary-somatomedin axis in patients with panic disorder is warranted given recent observations that several different stimuli of GH release fail to produce normal levels of GH in these patients (11).

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Book Forum

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MIND AND BODY

Psychiatry and Biological Factors, edited by Edouard Kurstak. New York, Plenum, 1991, 306 pp., \$55.00.

This book contains 26 chapters by a mixed culture of immunologists, epidemiologists, virologists, brain imagers, and stress researchers. They are united by an interest in the idea that viral infections may be major causes of mental illness, an idea which has long attracted the same sort of passions as does belief in unidentified flying objects. That being the case, it is strange that the book's title makes no mention of either viruses or immunology.

Three forces have recently reinvigorated the field and attracted reputable scientists to the Montreal conference on which this book is based: 1) the rise of AIDS and its neuropsychiatric consequences, 2) the epidemiologic evidence supporting prenatal viral theories of schizophrenia, and 3) the acrimonious public debate concerning the origins of the chronic fatigue syndrome.

AIDS is allotted two chapters in this book, but they are peripheral to its main theme. However, a number of contributors examine retroviruses and their relevance to schizophrenia; the answer seems to be none. Indeed, one feels rather sorry for those researching the virogene theory because its initiator, Tim Crow, makes it clear in the very first chapter that he has abandoned it. This theory seems to have been a temporary resting place on his voyage from believing that schizophrenia is wholly environmental to a belief that it is wholly genetic.

Different chapters examine psychiatric patients for abnormal rates of viral antibodies (especially herpes simplex and cytomegalovirus), immunoglobulins, T lymphocytes, and immunomodulators such as interferons. Several claim to have found immune dysfunction, but these are outnumbered by sounder studies with either negative findings or evidence that the positive findings are secondary to neuroleptic medication.

The high spot is a review by Mednick and colleagues of their important work from Helsinki and Denmark showing that exposure to influenza epidemics during mid-gestation increases the risk of later schizophrenia. There follows a somewhat apologetic chapter by Torrey, who failed to find any effect of the 1957 influenza epidemic on schizophrenic births in the United States. He gives an unusually frank account of the deficiencies in his study. Both of these chapters were written before confirmations of Mednick's results started rolling in, and there is little in the book to explain why prenatal influenza should be "schizophrenogenic."

It is a relief to turn from researchers furiously trying to implicate viruses in psychiatric conditions to one attempting the reverse. Simon Wessely attacks the idea that the "postviral fatigue syndrome" is a consequence of viral infection. Instead, he concludes that the majority of patients with this syndrome, who tend to be diagnosed as having myalgic encephalomyelitis in Great Britain and chronic Epstein-Barr virus infection in the United States, have a primary psychiatric disturbance. Thus, the moral of this book appears to be that viruses are not in-

volved in a disease in which most doctors think they are implicated—"postviral fatigue syndrome"—but are probably involved in a disease in which most psychiatrists think they are not—schizophrenia. Curiouser and curiouser.

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Memory in Mind and Brain: What Dream Imagery Reveals, by Morton F. Reiser. New York, Basic Books, 1991, 211 pp., \$27.95.

Strange but evocative, intensely personal but universal, dreams challenge us. Variably, dreams have been viewed as portents of future reality, carriers of universal symbols, random sensory images of the brain, and meaningful expressions of the sleeper. In *Memory in Mind and Brain*, Morton Reiser draws extensively from both psychoanalytic studies and current findings in neurobiology and cognitive neuroscience to examine the nature of memory and dreams. In short, he advances a contemporary psychobiological theory of dreams. An analyst himself, he uses a "dual track" approach whereby a phenomenon (specifically, memory in dreams) is examined from two theoretical perspectives, psychodynamic psychology and neuroscience, and then similar or isomorphic concepts from each theoretical perspective are combined to develop a comprehensive model.

The book is well organized, divided into three sections. The first section outlines the contributions of contemporary psychoanalysis to the understanding of memory and dreams. The presentation is concise and includes relevant case material. The second section reviews the current knowledge from neurobiology and cognitive neuroscience on the mechanisms of memory and sleep. This complex material is presented with clarity and helpful diagrams. In the third section, the understandings and knowledge from these two diverse fields are combined to form a new model of memory in dreams.

What does contemporary psychoanalysis contribute to the understanding of memory in dreams? By reexamining Freud's dream of the botanical monograph and dreams from his own case studies, Reiser develops Freud's notion of "nodal images." Dream images and the associations to these images are linked by the capacity to involve or evoke the same emotion. It appears that sensory residues in the mind are arranged in memory networks that are organized by affect. The organization of memories in dreams into networks linked by specific emotions helps explain two phenomena known to occur in psychoanalytic work with dream material: 1) "the memories of experiences involving the same emotion can be represented by an image connected with one of them (a nodal image)," a process Freud termed "condensation," and 2) the deeper meanings and memories (latent content) can be derived from associations to a dream image (manifest content).

What does contemporary neuroscience contribute to the understanding of the nature of memory and dreaming? The

work here is extensive. Recent studies in visual processing show that different aspects of a retinal image are processed by different neural pathways and in distinct cortical regions. Some cortical regions have extensive connections to the amygdala and the hippocampus, both of which are important to associative memory and emotions. Models of memory from neuroscience support the concept that memories of experience are both stored and recalled in networks of neurons that include not only the sensory but also the emotive aspects of the experience.

Seeing an overlap in the observations on the nature of memory from these two theoretical perspectives, especially that memories appear to be stored in networks linked by the same emotion, Reiser synthesizes a psychobiological model of memory in dreams. This model incorporates information from the neurobiology of sleep, visual sensory processing, and memory as well as from psychology, including the impact of current life stresses and conflict and their association to past experience. This model, a synthesis of psychodynamic psychology and neurobiology, represents a formidable challenge to models of dreams that are untheoretical. The author illustrates both the scientific advantage and the conceptual necessity for interdisciplinary work in order to advance the understanding of complex mind-brain phenomena.

The main weakness of the book is the failure to place or orient the presentation within the current debates on the philosophical problems that arise with the intertheoretical comparison or integration of terms and concepts from neuroscience and psychology. I also think that the concepts of affect and emotion, the linchpins in this model integrating the neurobiology and psychology of dreams, need a fuller presentation and theoretical grounding.

Despite these limitations, the book is highly readable and informative and is worthwhile for the psychologist interested in the neurobiological aspects of dreams and memory, the neuroscientist interested in the psychological aspects of memory in dreams, and the clinician or theorist challenged by a comprehensive model of mind-brain function in the phenomenon of dreams.

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SCHIZOPHRENIA

The Concept of Schizophrenia: Historical Perspectives, edited by John G. Howells, M.D. Washington, D.C., American Psychiatric Press, 1991, 201 pp., \$30.00.

This book elicited in me a strong sense of ambivalence regarding the progress made in the field of schizophrenia over the years. Reviewing the history of the concept of schizophrenia shows us how much we have indeed progressed, how much clearer the definition of the disorder is, and how much more agreement we have reached. However, it also provides a certain sense of stagnation, a sense of repetitive cycles of definitions of the concept, and a sense of humility about how little farther we find ourselves today than we were in Kraepelin's time. Over the past 100 years the concept of schizophrenia has experienced first a narrow stage, then a substantial widening, and now a move again toward a narrowing. Although there have been changes and evolutions in the concept through these years, as manifested by the recent introduction of a syndromal approach and the emergence of a wealth of new biological

correlates of the disorder, one feels that the field is ready for a new conceptualization, a new paradigm that could reorder phenomenology, biology, and course of the disorder in a radically new set of directions.

In the meantime, this book traces quite admirably the historical development of the concept with the aim of leading up to ever more precise descriptions and concepts of the disorder—a *conditio sine qua non* for any successful research effort.

Clearly, the disorder was recognized in Graeco-Roman times. Hippocrates described hebephrenic and catatonic states. Galen quite accurately reported on negative symptoms as being part of the disorder. Their pathogenetic concepts were expressed in the prevailing theory of psychic life of the times. Most of the time these forms of madness (mental illness) were clearly separated by early scholars from cases of mental retardation. In fact, there appears to be an early trail of clinical concepts that describe states of dementia appearing in adolescence. The description by Aetius of Amida (sixth century A.D.), of "those affected by fatuitas" as "young people with modest but intact mind, who after the disease appear as though demented" clearly is an early precursor of dementia praecox.

The chapter that describes these early historical descriptions covers well the Graeco-Roman-Byzantine-Arab period. It highlights the many surprising continuities between the ancient and modern conceptualizations of schizophrenia, such as the predominance of negative symptoms, the early age at onset, the disturbance in thinking, and the catatonic features. Additionally, one would have liked to see an exploration of early descriptions of schizophrenia in other cultures, such as the Indian or Chinese cultures.

Descriptions of mentally ill patients during medieval times remain diagnostically ambiguous, giving rise to the differential diagnosis of schizophrenia, psychotic depression, and mania. Finally, a clinical description that allows a more certain diagnosis of schizophrenia was provided by John Haslam in 1809. The chapter on nineteenth century developments highlights the contributions of Hecker, Bayle, Kahlbaum, and Morel in laying the foundations for the modern conceptualizations of schizophrenia. The description of the German classical concept of schizophrenia spans the two opposing poles of Kraepelin and Schneider with the latter's emphasis on positive symptoms and consequent widening of the concept. This chapter is counterbalanced by an excellent summary of Jaspers' contribution to the concept of schizophrenia, which emphasizes his reintroduction of the subjective to the study of psychopathology in general and of schizophrenia in particular. In the further development of the European views of schizophrenia, the Nordic school's contributions are almost completely missing, such as Leonhard's addition of the concept of the "third psychosis" with full recovery.

The American concept of schizophrenia is well described and traced back to both Bleuler's influence and the influence of psychoanalysis. These resulted in an extreme broadening of the concept, best expressed in the vague diagnostic criteria for schizophrenia in DSM-II. The chapter on American concepts retraces the correction of the pendulum swing in American psychiatry with the elaboration of several newer operationalized diagnostic systems for schizophrenia in the 1970s and 1980s. Unfortunately, more recent developments are not included, such as Crow's type I and type II schizophrenia, a serious shortcoming.

A short but excellent chapter by Bender on the concept of childhood schizophrenia follows and forms somewhat of a

bridge to the remaining four chapters, which cover psychodynamic and family concepts of schizophrenia.

Stone contributes two well-articulated and detailed chapters on the psychodynamic concepts of schizophrenia. He presents a model in which traditional formulations are integrated with modern neurophysiological data. He stipulates "distal etiological" factors such as genetic abnormalities underlying hedonic dyscontrol and cognitive impairments. The timing of decompensation, particularly for the young schizophrenic patient, is still seen as dictated by developmental conflicts. "Defect thus antedates but is influenced by conflict" (p. 164).

The chapter on family psychopathology and schizophrenia is one of the best chapters of its kind. It spells out by which criteria these concepts must be judged for establishing their scientific validity. Then each school of thought is critically reviewed in terms of its sufficiency regarding these criteria. The review finds most of the family systems schools wanting in terms of their conceptual formulations of schizophrenia. Unfortunately, this chapter does not include a review of the expressed emotion literature, which has replaced somewhat the more traditional family systems conceptualizations of schizophrenia.

The final chapter, although interesting, does not fit into this book at all, and it is not quite clear why it was included. It describes the work of two dedicated psychoanalytically oriented psychiatrists and their treatment of schizophrenic patients in the 1920s.

Overall, this book has many strengths and weaknesses. Although it is somewhat uneven in the representations of the cultural historical perspectives chosen, it does convey a solid review of the Western historical development of the concept of this disorder. Features of schizophrenia are clearly recognizable in the earliest descriptions of the syndrome. Certain manifestations may have changed over the centuries, but it appears that the complex of negative symptoms has been recognized as a constant feature. The organic nature of schizophrenia is another thread running through its history up to the present time. Reading about this past history will help every modern student of schizophrenia better understand the complexities of the disorder.

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PHARMACOTHERAPY

Integrating Pharmacotherapy and Psychotherapy, edited by Bernard D. Beitman, M.D., and Gerald L. Klerman, M.D. Washington, D.C., American Psychiatric Press, 1991, 445 pp., \$48.95.

Many of us who embrace eclecticism approach a work of this sort with a skepticism born in the experience of psychoanalysis and the influence of Carl Rogers, the existentialists, and others who urge an intensely personal therapeutic model, as well as the broadening importance of the biologists and pharmacologists in psychiatry. The stridency of highly visible proponents of exclusionary models deepens concerns that integration will not work, that a "rating scale/checklist" mechanistic formulation will replace the close patient-centered therapeutic tradition, and that no one except the insurers and regulators will be happier or better off for it. Nonetheless, the premise of this carefully selected and edited text—the integration of two seemingly disparate models of therapy, the rap-

prochement between alien professional camps, a synthesis of science and humane experience in the service of the individual patient—is well developed and demonstrated, to the extent that reconciliation ultimately appears not only possible but inevitable.

Churchill's description of American and British cultures divided by a common language resonates fittingly in our profession. We continue to be divided by our sense of how the mind works and how to best intervene therapeutically when the mind works poorly. Although growing third-party intrusion is reducing the number of us who can afford to be therapeutic "purists," there remains discomfort in integration. The late Gerald Klerman is probably identified more strongly than any other prominent psychiatrist as a reconciler of our differences. In this book, with contributions and leadership by Drs. Klerman and Beitman, 33 highly qualified clinical scientists promulgate their ideas and methods in 22 chapters within four sections, entitled Ideology and Process, Clinical Implications of Research Into Specific Diagnoses, Other Diagnostic Considerations, and Speculations.

Klerman wrote the first chapter, presenting the ideological underpinning of the book and a summary of his working philosophy in this area. The material will be familiar to anyone who has been exposed to Dr. Klerman's work, but here it serves to recount why a book like this is necessary at all. If chiropractors were within the ranks of orthopedists, we might see among surgeons the sort of practical and philosophical division that permeates psychiatry. Alas, however, the depth and breadth of our internal antagonisms are essentially unique in organized medicine.

The second chapter, by Dr. Beitman, proposes a four-stage model for integrative individual psychotherapy. A series of clinical vignettes illustrates some of the points of emphasis: cases involving medications as engagement techniques, ineffective medications as engagement, and transference in the engagement stage illustrate three of the many vignettes Beitman uses to elucidate each stage of the model. They are frustratingly brief, however, and therefore limited in their usefulness; an entire book could properly address these matters.

Chapter 3, "Diagnosis-Specific Psychotherapy," has lots of good advice for recognizing individual patient characteristics in planning treatment, maintaining flexibility in changing diagnostic impressions and therapeutic approaches, and rejecting dogma in selecting treatments (which means, of course, combining medication with verbal therapy). These are suggestions of psychotherapists who are approaching pharmacotherapy in practice. It is a good paper, but I remain a little puzzled by just what "diagnosis-specific psychotherapy" is and how to reconcile the chapter content with the title.

Chapter 4, "Psychosocial Approaches to Pharmacotherapy," says many of the same things (careful diagnosis, attention to the individual, and guidelines for combination therapy) but is presented from the perspective of a clinical pharmacologist advocating a role for psychotherapy. A section on special problem patients includes a fairly complete case history of a hostile-dependent patient as well as briefer but sufficient paragraphs on special problems of patients with narcissistic, obsessive-compulsive, borderline, and substance abuse disorders. It also has an interesting portion on the "natural, holistic patient," which deals with the arguments regarding the efficacy of unorthodox or nontraditional methods such as vitamins, allergy prevention, and crystals. I think this is one of the best sections of the book, with pithy and abundant detail from an experienced and thoughtful teacher.

The second section, written by currently active investigators, concentrates on specific considerations of most DSM-III-R

diagnoses. The authors quote extensively from their own work and survey the research of other authorities, suggest interpretive guidelines, and provide a thorough bibliographic listing in each chapter. There are some stretches that are already dated (the work in this 1991 publication must have been done in 1989 or 1990), but most of the material is sufficiently solid to remain unaffected by the inevitable lag between preparation and readership.

The remaining two sections contain five chapters that thematically and substantively extend the process of seeking common clinical and philosophical ground by means of illuminative research reports and therapeutic recommendations. In "Interpersonal Psychotherapy for Depression," Weissman and Klerman discuss how major depression usually occurs in an interpersonal context (an interpersonal loss or dispute) susceptible to the process of clarifying, refocusing, and renegotiating issues that can occur in interpersonal therapy, accelerating recovery and reducing social morbidity. Regina Casper's chapter on outpatient treatment of anorexia nervosa explores the use of psychotherapy, medication (antipsychotics, lithium, and pro-peristaltic agents), and hormones. It includes an illustrative vignette and notes emphatically that no drug can reverse the severely ill patient's fear, panic, and fixation on thinness. Kay Jamison's chapter emphasizing the need for psychotherapy in the treatment of manic-depressive illness will resonate with anyone concerned that the so-called lithium clinics too often devalue this vital aspect of therapy.

"Contributions to the Development and Treatment of Panic Disorder: Toward a Piece of Mind and Brain" by Eric M. Reiman and "Exposure and Desensitization as Common Change Processes in Pharmacotherapy and Psychotherapy" by Drs. Beitman and Mooney are, in many ways, reprisals and re-explorations of earlier thematic material, tending to concentrate the central issues of the book as it approaches its conclusion.

I highly recommend this book to my fellow eclecticists and to those who have been otherwise troubled by psychiatry's discontinuities. Drs. Beitman and Klerman have done a particularly good job of transcending the annoying and familiar problems of the multiauthored collection—repetition, cross purposes, and absence of a defining structure with clear conclusions. This book moves toward a conceivable vision of our specialty's future, with the intellectual excellence of each faction applied to the relief of diseases that do not honor our boundaries. Published as Dr. Klerman's outstanding career was coming to an end, this volume is a testimonial to his work as an integrator and a signal of further advances by his colleague and protégé, Dr. Beitman.

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Pharmacotherapy of Depression: Applications for the Outpatient Practitioner, edited by Jay D. Amsterdam. New York, Marcel Dekker, 1990, 461 pp., \$79.50.

A few years ago a book publisher said to me, "There are too many books on depression; another one won't sell." Indeed, there was a time when numerous research volumes on biological factors in depression seemed to fill up library shelves at an alarming rate. However, the great majority of those books were not for the clinician; in truth, they contained the reports of contradictory studies, many of which have not been successfully replicated.

The present book represents a maturing of biological psy-

chiatry. The audience is clearly the general psychiatrist; no attempt is made to woo him or her with recommendations to measure urinary 3-methoxy-4-hydroxyphenylglycol or CSF 5-hydroxyindoleacetic acid; the book has a generally modest, useful tone. No apologies are made for restricting the book to pharmacotherapy. There have not been too many books on depression in the last 5 years, since the focus of biological research moved toward obsessive-compulsive disorder and panic disorder. Therefore, the present volume is a useful and balanced update for the clinician.

The title of the volume contains the word "outpatient," but there is little in the volume aimed specifically at outpatient rather than inpatient treatment. The material is specialized and directed at psychiatrists rather than family physicians, who treat most outpatient depression. There is very little attempt in the book to deal with the central question of outpatient depression practice, namely, do the large number of atypical, nonendogenous, masked, neurotic, and characterological depressions respond to pharmacotherapy better than to placebo? Such a question is especially central to a book that limits itself, in its title, to pharmacotherapy.

On page 2 we read that 20%–26% of women and 8%–12% of men will suffer from major depression at some time in their lives. On page 75 we read that the response of depressed patients with neurotic, hypochondriacal, or hysterical personality traits to tricyclic antidepressants is no better than it is to placebo. Nowhere in this volume, and perhaps nowhere in our present scientific database, can we learn whether the criteria for major depression in epidemiologic studies is congruent with criteria for "endogenomorphic" (p. 76) depression in pharmacological studies. It can be argued that the epidemiologic criteria, by necessity usually retrospective and/or standardized and administered by nonpsychiatrists, are much broader than the criteria used in controlled drug trials of well-studied small numbers of patients. Thus, much of the "depression" seen by outpatient practitioners may not be drug responsive, and it would be questionable today to exclude cognitive and family therapy from any textbook on the treatment of depression. Indeed, a chapter that detailed the indications for referral for psychotherapy would be a minimum even in a volume on pharmacotherapy. Cognitive therapy, family therapy, marital therapy, and psychotherapy do not even appear in the index to this volume.

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Medicine and Mental Illness: The Use of Drugs in Psychiatry, by Marvin E. Lickey and Barbara Gordon. New York, W.H. Freeman & Co., 1991, 443 pp., \$21.95 (paper).

Even after several decades of revolutionary advances in diagnosis and treatment in psychiatry, many cannot accept that mental illness can be due to biochemical changes in the brain and that drugs have a proper role in the treatment of mental illness. It is not surprising for a psychiatrist to come across questions like, Do you really treat the mentally ill with drugs? or We have a violent patient; why don't you dope him? even from medical students and other physicians. Educating people is still a major task of the psychiatric community. *Medicine and Mental Illness* is a welcome addition to the few books in this regard.

This book is organized into four major parts. In the introductory chapters the authors briefly explain about the introduction of drugs into psychiatry and discuss related issues. A

clear and well-illustrated biochemical account of how drugs act in the brain is followed by a chapter on the evolution of modern diagnostic procedures and DSM-III-R. The rest of the book deals with schizophrenia, affective disorders, and anxiety disorders, with a concluding chapter on the medical model of mental illness.

The syndromes are presented under the headings of diagnosis, pathophysiology, treatment, and adverse reactions to drugs. The discussions of the respective diagnoses are excellent, explaining each criterion in detail with case examples. The recent developments in the understanding of pathophysiology are well reviewed with extensive references to the studies on the subject. In discussing treatment, the authors cite the research literature and explain the methodologies and outcomes in detail. The mechanism of action of drugs and their adverse effects are thoroughly discussed with very good illustrations. These chapters will answer any question regarding the usefulness of drugs in psychiatry with authenticity. Although the book is on drugs in mental illness, the importance of psychotherapy is also highlighted in appropriate places. The authors weigh the benefits of drugs over side effects and discuss other modes of therapy such as ECT, behavioral therapy, psychotherapy, and combinations of these for individual cases. The concluding chapter is very impressive; here controversies regarding biological and psychological theories, criticisms, and ethical issues are addressed.

Written for the scientific layperson, the book largely avoids or explains technical language; however, the presentation is very scientific and authoritative. This book is easy to read and is a source of quick reference, particularly for biological aspects and mechanism of action of drugs. It has a well-compiled bibliography. This book would be very useful for anyone who needs to understand the role of drugs in psychiatry and their biological basis. This book is highly recommended for students, trainees, and physicians who have no formal training in psychiatry.

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Mental Retardation: Developing Pharmacotherapies, edited by John J. Ratey, M.D. Washington, D.C., American Psychiatric Press, 1991, 162 pp., \$29.50.

This small volume is part of the Progress in Psychiatry series published by the American Psychiatric Press. The fact that this publisher decided to produce a book dealing with the treatment of the mentally retarded and mentally ill individual is cause for some to be encouraged. That this effort is so uneven is disappointing. The book does not live up to its cover notes in that it is not a "practical guide to the treatment of the developmentally disabled." Perhaps the editor and the readers would be better served if it were not advertised as anything more than what it is: a series of essays, some extremely pertinent, by some of the leading writers in the field.

Any text that deals in developing pharmacotherapies, especially in the area of developmental disabilities and mental illness, runs the risk of being dated or argumentative before the ink is dry on the page. This book suffers that fate as well. The work is also weakened in that some of the chapters are misplaced in this volume due to their theoretical viewpoint or lack of relevance to clinicians. This is not to say that the work is without merit, only that it is a disappointment.

An example of the type of paper included in the book is the first chapter, "Neuropsychiatry and Mental Retardation,"

written by the editor and C. Thomas Gualtieri. Both are widely read and respected in this field. They discuss several topics briefly, including the philosophical underpinnings of psychiatry, their theory on why psychiatry has "retreated" from the developmentally disabled (for an interesting counterpoint on specialization and educating psychiatrists read Herb Pardes' 1989 article [1]), learning theorists, the medical models, and the "new age" of neuropsychiatry. Topics of great interest and timeliness include "noise," stress and aggression, "kindling," and the environment in which psychopharmacology and social interactions exist.

There is much that is worthwhile in this book by important writers and clinicians, and the topic of psychiatric treatment for mentally retarded individuals is painfully underrepresented in publishing lists and residency training programs. It is too bad that this work does not have more to recommend it.

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Human Psychopharmacology, vol. 3: Measures and Methods, edited by I. Hindmarch and P.D. Stonier. New York, John Wiley & Sons, 1991, 224 pp., \$96.00.

By identifying their topic as "human psychopharmacology," the editors of this series of monographs give notice of the broad territory they intend to survey. Volume 3 meets these expectations, with a broad sampling of the spectrum in a rapidly expanding and diverse field.

Each chapter in this multiauthored volume represents a free-standing review of a current research technology within the domain of clinical psychopharmacology. Individual chapters are not clustered into thematically organized units, and there is little to suggest that the editors intended this as a textbook. As the title implies, the monograph will be of more immediate relevance to the clinical researcher than to the practicing clinician. The investigator with experience in a specific methodology may find the chapter on his or her area somewhat generalized, however.

Given the diversity of topics covered in this volume, there is likely to be something for everyone: topographic EEG, critical flicker fusion threshold, eye movement patterns, sleep patterns, long-term clinical trials with antidepressants, psychobiology of the menstrual cycle, analgesic effects of psychotropic medications, compliance in pharmacological trials, risk assessment in clinical trials, cardiovascular effects of psychotropic medications, psychomotor effects of alcohol, and epidemiology of substance abuse.

Individual chapters in the book appear to reflect consistently high levels of scholarship and include extensive bibliographies. Editorial style across chapters is relatively consistent for a multiauthored volume. There is a notable degree of variability, however, in the extent to which individual chapters focus on details of methodology versus summaries of recent clinical findings based on application of the methodology.

In summary, this volume represents a highly useful reference resource for the reader setting out to explore the technologies of human psychopharmacology.

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PSYCHOTHERAPY

What Is Effective in Psychoanalytic Therapy: The Move From Interpretation to Relation, by William W. Meissner. Northvale, N.J., Jason Aronson, 1991, 216 pp., \$32.50.

Fully a quarter of this densely written, almost inaccessible, slim volume is devoted to a reprint of James Strachey's 1934 paper: "The Nature of Change in Psychoanalysis." Meissner then recapitulates Strachey's argument. Strachey argued that before 1934 Freud and his circle thought that psychoanalysis changed people by bringing drive-related information out of repression and into consciousness. However, they realized that when resistance prevented emergence of such material, the growing relationship between doctor and patient became an important part of this process. Analysis of the inevitable distortions in this dyadic interaction then became the central focus of interpretation. Therapeutic effectiveness was linked both to the mutative power of interpretations aimed at points of urgency that were therefore thought to be id-related and to the fact that the patient grew less self-punitive as the analyst, functioning as a new ego ideal, became incorporated into the patient's superego.

In the succeeding 140 pages of the largest type and widest interlinear spacing I have seen in a contemporary book, Meissner proceeds to summarize the ways psychoanalysis has changed in the 60 years since Strachey's contribution. He nods at ego psychology, object relations theory, self psychology, and the more recent shifts in developmental theory. The author's theme is evident early: all of our theories lead inevitably to a renewed focus on the therapeutic alliance, on the importance of the relationship (both real and transference) that evolves during the analytic process. The growing interaction between analyst and the observing ego of the patient is defined as part of the therapeutic alliance. Whatever "facts" are brought out during the analytic process are shown to be distorted by the theories most important to the analyst.

The central concept of this book is important and well stated:

The translation of the patient's inner psychic processes into verbal terms is paralleled by a similar translation that takes place within the analyst. The associative basis includes the associative material that arises within the analyst's consciousness during the analytic process. While such data are in a sense observable, they are better regarded as introspective The important point is not that the nature of the data is any different whether it arises in the patient or in the analyst, but that the inclusion of data coming from the analyst creates a broader data base that includes mental processes taking place within two interacting individuals. The patient's verbal behavior is not merely an objectified observable phenomenon; it is at the same time a process of communication from a subject to an object. The patient not only speaks, but speaks to another person, specifically the analyst. The speaker does not speak *in vacuo*, but addresses a listener. And both are engaged in a dialogue of mutual speaking and listening. The observational model has a bias built into it that isolates the data of observation into the object of observation. The present consideration would seem to extend the proper data base of psychoanalysis beyond the limits of a strictly observational model. (pp. 87, 88).

The remainder of this short, dense book is devoted to a reinterpretation of psychoanalytic theory in terms of this comment. The difference between historical and narrative truth is discussed, as is the resultant shift in our understanding of the art of genetic reconstruction. Meissner concludes with a look at the concept of empathy and the significance attributed to it by the various schools of psychoanalysis. How and why empathy and relatedness cause structural change remain just as mysterious as the magic that makes an interpretation mutative.

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Comparing Psychoanalytic Psychotherapies: Developmental, Self, and Object Relations, Self Psychology, Short-Term Dynamic, by James F. Masterson, M.D., Marian Tolpin, M.D., and Peter Sifneos, M.D. New York, Brunner/Mazel, 1991, 298 pp., \$39.95.

In an article published in 1975 (1) James Masterson and Donald Rinsley described their views about the role of the mother in the development of individuals with borderline personality. Masterson and Rinsley believed, on the basis of their own observations and theory, that the withdrawal of maternal support when the child attempts to assert itself to progress toward separation and individuation specifically results in borderline development.

Both Masterson and Rinsley elaborated and modulated their views in numerous subsequent publications (2, 3). Masterson's *Psychotherapy of the Borderline Adult—A Developmental Approach* (3), published in 1976, is in its tenth printing, with more than 50,000 copies in print; it has been translated into German and Japanese. Masterson is the author or coauthor of at least nine volumes on borderline and narcissistic disorders; one of these (4) was recommended by Gloria Steinem as one of the "books that I've found mind-opening myself or that I've witnessed as rescuing for others" in an appendix entitled *Bibliotherapy* in her most recent bestseller (5).

These details signify the popularity and influence of Masterson's views and the profoundly felt need his elaborate treatment approach to patients with severe personality disorders fills. Masterson's seminal contributions demonstrate his unswerving seriousness, tireless experimentation, and complex optimism in treating a great variety of people with what we call, for lack of a better term, borderline disorders. Masterson is the founder of an institute bearing his name that is devoted to both the treatment of borderline and narcissistic disorders and to the teaching of his approach to these disorders.

Comparing Psychoanalytic Psychotherapies is the printed record of two conferences organized by the Masterson Institute in New York and San Francisco, respectively, in February and March 1990. The conferences had two purposes. The first was to contrast the treatment of people with severe personality disorders by two major psychoanalytic psychotherapeutic processes—the developmental, self, and object relations method (Masterson's) versus the self psychological approach, originally developed solely for the treatment of narcissistic personality disorders by the late Heinz Kohut (6, 7). The second purpose was to differentiate both of these procedures from short-term dynamic psychotherapy.

The framework of the book follows that of the conferences. First, three patients treated by members of the Masterson Institute are described: a patient with lower-level borderline per-

sonality disorder is presented by Shelley Barlas Nagel, Ph.D., a patient with narcissistic personality disorder is presented by Ralph Klein, M.D., and a patient with borderline personality disorder is presented by Karla Clark, Ph.D. The intervention with the patient with lower-level borderline disorder illustrates Masterson's supportive therapy, with the limited objective of some "ego-repair," and the treatment of the patients with narcissistic personality disorder and borderline personality disorder are described as three-times-a-week, long-term, intensive psychotherapies, geared to ultimate cure. Each case is followed by discussions by the three authors of this volume, including questions raised by the conference participants and the authors' responses. Each of the three authors then conducts a workshop on his or her approach to psychoanalytic psychotherapy and fields more questions from the conference participants.

James Masterson comments from the vantage point of his treatment method. Marian Tolpin, distinguished co-worker and coauthor of Heinz Kohut, illuminates the discussion from the viewpoint of self psychology. Peter Sifneos, pioneer of short-term anxiety-provoking psychotherapy, highlights the therapeutic needs of patients with severe personality disorders by way of discussing patient selection criteria for short-term anxiety-provoking psychotherapy and its limitations. Sifneos' participation adds significantly to the stimulus of the discussion both because of his good humor in sharing his rocky start as a candidate at the Boston Psychoanalytic Institute while advocating short-term psychotherapy and because of his expertise in sharply defining the kind of people and problems amenable to short-term psychotherapy. Sifneos' criteria eliminate the vast majority of, if not all, borderline and narcissistic patients from consideration.

Of course, the developmental theories of Tolpin and of Masterson are critically different. A fundamental tenet of self psychology is that the emerging self requires objects to provide experiences that will facilitate further development of the self. These "selfobject" experiences invigorate the self and lead to transmuting internalizations, in the course of which aspects of the selfobject experiences are internalized and, as it were, metabolized to be used as building blocks for structuring a more cohesive self. Hence, the fundamental precept of self psychology, the transmuting internalization of selfobject experiences, is very different from that of object relations theory, i.e., the internalization of self and object representations, whether as whole-object relationship units in normal development or as part-object relationship units in borderline and narcissistic pathology (3, 8, 9).

Tolpin points out, "I feel that I have a big job here" (p. 21). "You've got to understand the differences in point of view before you start talking about this or that intervention. It's like starting to play a board game and not knowing any of the rules. The opening moves, as it were, belong to the basic orientation of the therapist" (p. 32). "We're not going to get a divorce because we've never been married, but we're going to go on struggling to communicate. There are people who think it's hopeless to try to translate from one theoretical framework into another. I am not one of them" (p. 36). Thus, in discussing a borderline patient Tolpin feels that the selfobject transference should be placed at the center of the treatment and understood in terms of its disruptions, but Masterson maintains that "you have to view the ego structure of the borderline as similar to a piece of Swiss cheese with all these holes in it in terms of reality perception . . . What the therapist does by confrontation is lend the patient his/her reality-testing capacity. The patient identifies and internalizes this . . . The task

of treatment, as we see it, is to convert transference acting out into therapeutic alliance and transference" (pp. 19-20).

These brief exchanges give only a glimpse of the energy, conviction, debating skills, and dexterity with which the three discussants put their ideas across. I knew early on that I was going to like this book when I first came to Masterson's and Tolpin's sallies. Their discussions are enormously helpful. Beyond the bare outlines of discontinuous theories in individual books, these confrontations give the reader a front-row seat to Masterson and Tolpin, as it were, supervising each other in psychoanalytic psychotherapy. Masterson argues with relish and gusto; he is assertive, even aggressive, and sounds a bit strident at times in promoting his valid and provocative insights. Tolpin puts her points across with considerable verve and verbal elegance and is very sophisticated both clinically and philosophically. Her remarks are deliberate, intensely attuned, and steadfast in her discussion of each patient: "There is no diagnosis in dynamic psychoanalytically oriented psychotherapy that can be made from a cluster of symptoms that does not do you and the patient an injustice. You do it as you go along and you have to think about it" (p. 38).

Tolpin peels away the seductive web of habitual words and makes sure that boundaries do not get obscured in the ebb and flow of clinical discussion: "I can see from this case history why there's a flirtation. We seem to be talking the same language and then suddenly we're not . . . And then the flirtation ends in trouble because we're going down different theoretical outlooks that have enormously affected what is considered defense, what is considered the primary pathology, and how you intervene with it" (p. 53).

Of course, the truth of the matter is, as Tolpin says, "No psychoanalytical theory is quite right, no theory is quite wrong. Each is a particular way of looking at things in a particular era. We decide about which theory, I think, on the basis of how attractive it is to us, personally. How much can we understand it? How useful is it clinically? Psychoanalysis is not a hard science so that you have to try to recognize what the choices are that go into the whole banquet that's around you from which to choose" (p. 231). For many severe personality disorders, Masterson's (and Kernberg's) confrontational approach is clinically very efficacious. For patients with higher-level borderline and narcissistic disorders, the self psychological approach advocated by Tolpin, elaborated brilliantly and systematically by Adler (10), is suitable as well, perhaps preferable. Despite irreconcilable metapsychological differences, on purely clinical grounds, both approaches have much to recommend themselves with different patients and different therapists. Particularly with this group of difficult patients, flexibility is important so that the most appropriate treatment modality can be selected from a range of potential psychotherapeutic interventions.

I have two caveats. First, "borderline" as used in these discussions does not always refer to the narrow definition of borderline personality disorder of DSM-III-R. Rather, it reflects Kernberg's broader psychostructural category of borderline personality organization (8). Thus, what is specifically referred to as a high-level "borderline personality disorder" in this book (Karla Clark's case) does not meet the narrow diagnostic criteria set forth in DSM-III-R. Second, this volume is concerned solely with psychotherapeutic strategies. It does not broach the role of psychotropic drugs in the treatment of severe personality disorders even though at times psychotherapy is not possible with these challenging people without the adjuvant use, possibly temporary, of various classes of medications.

Need I add anything? Yes. I should add that psychiatrists

in the managed care industry would do well to study this book assiduously. Its analysis of the care and treatment needed by these very difficult patients is both penetrating and poignant as well as hardheaded.

In sum, this is a delightful, reader-friendly, incisive, instructive, useful, and generally optimistic volume. If you have considered getting and reading this book, my advice would be that you do so.

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Practicing Psychotherapy: A Casebook, by Michael Franz Basch. New York, Basic Books, 1992, 224 pp., \$30.00.

Tennis coach Vic Braden often told how, as a ball boy at the Davis Cup matches, he would watch the interaction between the greatest players of their era and Jack Kramer, surely the greatest coach in the history of the game. Braden wondered what Kramer mumbled to Arthur Ashe and Clark Graebner as they passed him at the change in courts. Did he, perhaps, say something befitting the advanced status of these champions ("The wind is 12 mph north by northeast. Hit 5 degrees away from the corner and the ball will drift in.")? Finally, Vic Braden got close enough to hear what this great coach said to these greatest of players: "Bend your knees. Keep your eyes on the ball."

Michael Franz Basch has written Jack Kramer's book of advice to excellent psychotherapists. Noting that the best among us gets stuck every once in a while, Basch suggests a way of conceptualizing the process of psychotherapy that is as embarrassingly simple, accurate, and useful as the core reminders in tennis. He asks us to forgo our usual focus on symptoms and work in terms of a developmental model based not on Freud's theory of psychosexual maturation but on the primacy of innate affect: "The common denominator that makes us human and holds us together is that we are born with a mobile face and a built-in information-generating program that readies us for affective responsiveness and affective communication."

Basch suggests that human life involves the search for competence and that only significant failure in this quest pushes

us toward the indignity of psychotherapy. Failure within a self-correcting system provides normal learning experience, but the kind of failure that results only in loss of self-esteem and further downward drift in competence can be traced to problems in early development. As infants, our roiling all-or-none affective responses are sensed by the caregiver and used as clues to the stimuli and needs that triggered them—Basch calls this the "kinship" experience. Soon the growing child learns that we adults can be counted on to handle the overwhelming situations that trigger affect—this realization encourages the child to idealize the caregivers. The growing sense that there is a connection between its expressed affect and parental attention to its needs offers validation of affective experience and further affective learning. Much of what brings the patient to psychotherapy can be traced to inadequacies in the parental system for kinship, idealization, and affective validation, and it is in these modes that the patient experiences us during much of the healing process.

Nowhere does Basch ignore the classical psychoanalytic approach to disorders traceable to mishaps in psychosexual development. This is not a manual designed to flog Freud and crown Kohut. It is a sensible description of the ways we can look at patients in such sectors of development as affect and cognition, attachment, autonomy, and creativity. For each cluster of problems there is a therapeutic approach that takes into account the place in the developmental line where failure occurred. Central to this book is the belief that there is an inherent urge toward wellness that can be accessed and amplified by intelligent treatment. Attention to "primitive" concerns leads not to "regression" but to growth; always, the road to effective treatment is found by meticulous attention to the patient's affect.

Do I make it seem that this a book of theory? Hardly. It is designed as a series of actual consultations between Basch and a number of therapists with a wide range of experience and skill. Particular attention is given to shame and anger, the negative affects that cause the most trouble in treatment. Although the ultimate focus of our work is the patient who comes for treatment, here we come to understand how therapeutic success is dependent on our own training and attitudes.

All of us get stuck every once in a while. Cases that seemed to be going well often get unaccountably mired in sessions like repeating loops of tape. The waggish comment that "a specialist is the guy who remembers the textbook" is nowhere more apt than in the art of psychotherapy. Sometimes we need a better textbook. Bend your knees. Keep your eyes on the ball. Follow the affect.

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ECT

The Psychiatric Clinics of North America, vol. 14, number 4: *Electroconvulsive Therapy*, edited by Charles H. Kellner. Philadelphia, W.B. Saunders Co., 1991, 228 pp., \$77.00.

This issue of *The Psychiatric Clinics of North America* devoted to ECT comes at a time when ECT has made a comeback in the treatment of psychiatric disorders. It is one of the most efficacious treatments we have, but it has been misperceived as invasive and brain damaging even though its efficacy is well proven. The misconceptions about ECT are due partly to its portrayal in the media as it was perceived in the past and partly

to the bias of the professional community and the antipsychiatry movement.

The book is organized into 13 chapters, each dealing with a different aspect of ECT, and has been written by experts in the field. Each chapter gives an exhaustive review of the literature pertaining to the topic being discussed and is followed by a good summary and recommendations. Each covers many areas of practical clinical importance in a style that would be particularly useful for ECT practitioners in their day-to-day practice.

In the first chapter, Max Fink gives the reader a succinct overview of the different reasons for resistance to ECT therapy in the community and poor funding in the area of research, even by large foundations that are well-known for supporting psychiatric research. The next chapter, by Harold Sackeim and colleagues, is an excellent discussion of the impact of stimulus intensity, seizure threshold, and seizure duration on and the efficacy and safety of ECT—all of extreme practical importance. This chapter includes a discussion of factors to be considered in daily ECT practice. The next chapter, by Richard Weiner and associates, on management and monitoring of electrically induced seizures, deals with toxicity and termination of prolonged seizures very well. ECT is a low-risk procedure, and Richard Abrams' chapter discusses in great detail its cardiovascular and cerebral effects as well as its use in the medically compromised patient.

The chapters on the use of ECT in mania and parkinsonism, by Joyce Small and associates and Keith Rasmussen and Richard Abrams, respectively, are of high quality and summarize the current concepts in this field. I was especially impressed by the contribution of Devanand and colleagues on the use of ECT in the treatment-resistant patient. They describe the controlled and uncontrolled studies to date on medication resistance and response to ECT. They also cover management of relapse and ECT-resistant depression. The chapters on treatment schedules and maintenance ECT, by Baruch Shapira and associates and Russell Monroe, are excellent overviews of the ongoing controversies in relation to the practice of ECT, such as the use of a twice-a-week schedule compared with three treatments a week in terms of their cognitive side effects and treatment response. The chapter on neuroendocrine testing by

Rifaat Kamil and Russell Joffe is mostly of academic importance.

ECT has become a multidisciplinary approach that involves nurses, psychiatrists, and anesthesiologists. This book has a good chapter pertaining to nursing care by Carol Burns and Gail Stuart, who emphasize the key role that nurses play as part of the treatment team. It was thoughtful to include chapters on choosing an ECT device (by Stephani Stephens and associates) and on the legal and ethical issues related to ECT (by Gregory Leong and Spencer Eth) to which many of us are oblivious.

This book covers almost all issues pertaining to state-of-the-art ECT, from informed consent to seizure production. It would have been prudent to have a chapter on pregnancy and ECT, which is of practical importance. The administration of anesthesia, which is mentioned in several chapters, should have been dealt with separately because there are still practitioners who do not use the services of an anesthesiologist. APA has published a task force report recommending guidelines for ECT (1), and these have been appropriately mentioned throughout the issue.

This book is easy to read, has an extensive bibliography at the end of each chapter, and provides complete information on several aspects of ECT. In addition, it is "user friendly," technically of high quality, well edited, and has few typographical errors and misspellings. It would be useful not only for ECT practitioners but also for all psychiatrists and residents in the field. ECT has made a strong comeback and should be taught in more residency programs because there is no controversy about its effectiveness, but hurdles related to stigma and public image remain to be completely conquered.

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Reprints of Book Forum reviews are not available.

Letters to the Editor

Extrapyramidal Symptoms and Cocaine Abuse

SIR: The early phase of cocaine abstinence, the "crash," has been linked to cocaine-induced disturbance in dopaminergic function (1, 2). Interestingly, cocaine does not seem to affect motor pathways in clinically observable ways. One might expect that motor symptoms (such as extrapyramidal symptoms) would be noted during the "crash" phase of cocaine abstinence, yet cocaine addicts tend not to report the type of muscle stiffness or bodily "heaviness" typically described by patients with parkinsonism or those medicated with neuroleptics. We report a case in which a cocaine abuser experienced extrapyramidal symptoms during cocaine early abstinence, or "crash."

Mr. A was a 40-year-old man with DSM-III-R diagnoses of cocaine dependence and generalized anxiety disorder predating cocaine abuse. Mr. A had free-based an average of 12 g per week of cocaine over the last 2 years. He was medicated with buspirone, 30 mg/day, for 1 month to reduce anxiety, but the trial was unsuccessful. Finally, thioridazine, 250 mg/day, partially relieved his anxiety. He displayed no signs of extrapyramidal symptoms.

Mr. A was admitted for treatment for addiction after a 2-day cocaine binge (7 g consumed). Urine toxicology screen revealed cocaine and phenothiazine. Routine chemistries, complete blood cell count, sedimentation rate, and creatine kinase were all within normal limits. Mr. A was most worried about the "muscle problems I always get when I crash—if you can't give me something for it, I'm signing out." He described classic stiffness and an overall weightiness to his body that generally dissipated by the time he awoke after 18–24 hours of postbinge hypersomnolence. He recalled that these symptoms occurred when he terminated a binge "ever since I started taking the pill" (i.e., thioridazine) but assumed it was a coincidence. Symptoms did not occur with thioridazine treatment in the absence of cocaine. During and immediately after "crashing" from cocaine (prior to treatment with a neuroleptic), Mr. A claimed he felt "physically exhausted, wiped out," but he explicitly denied muscle stiffness and weightiness.

Upon examination, there was mild cogwheeling at the elbow and diminished fluidity with passive arm and leg movement. This responded to amantadine, 100 mg t.i.d., which was prescribed for 48 hours.

Several lines of evidence demonstrate central dopamine reduction in animals exposed to repeated cocaine administration (3, 4). Neuroleptic drugs (e.g., haloperidol, thioridazine) also reduce dopaminergic transmission. Through dopamine receptor blockade in the nigrostriatal pathways these drugs can produce extrapyramidal symptoms including cogwheeling, rigidity, and akathisia. F.J. Gawin (personal communication) observed transient akathisia in five cocaine abusers after they received single neuroleptic doses as prophylaxis against cocaine-induced paranoia and violence.

Cocaine-induced motor effects may be subclinical unless unmasked during acute cocaine withdrawal or other instances

in which dopaminergic motor systems are vulnerable. Such examples could include neurological disorders, normal aging, prolonged pharmacological treatment (e.g., neuroleptic treatment, particularly in the context of cocaine-related paranoia), or neurotoxicity related to chronic stimulant abuse.

It is unknown whether perturbations of dopaminergic motor systems in cocaine abuse could amplify or accelerate subsequent expression of Parkinson's disease. This possibility indicates that individuals with new-onset movement disorders should be assessed for past history or ongoing use of cocaine.

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Heroin Withdrawal Precipitated by Nonmedical Use of Naltrexone

SIR: Naltrexone is a long half-life, orally active opioid antagonist (1). Developed as an aid to recovery for opioid addicts, clinical use of naltrexone requires care to avoid precipitated withdrawal (2). To date, neither naltrexone-precipitated withdrawal secondary to nonmedical administration nor severe withdrawal symptoms have been reported. Two cases are presented of naltrexone administration to heroin-dependent individuals in nonmedical settings.

Mr. A, a 38-year-old man with a 23-year history of intravenous heroin use, was regularly consuming 3 g (\$270 worth) of heroin per day in divided doses. Mr. A consumed 2 g of heroin over the course of a day, drank 64 oz of beer and 4 oz of whiskey, and injected 1 g of heroin. He collapsed and became apneic and cyanotic. His companions commenced mouth-to-mouth resuscitation and administered 25 mg of naltrexone intravenously. He rapidly regained consciousness and experienced dysphoria, hyperalgesia, nausea, vomiting, and diarrhea. Severe withdrawal symptoms lasted 8 hours with milder symptoms persisting for 3 days. Despite considerable apprehension, Mr. A was restarted on regular naltrexone therapy.

Mr. B, a 37-year-old man with an 8-year history of smoked heroin use, was smoking 0.1–0.2 g (\$80–\$160 worth) of

heroin in two divided doses per day. He ran out of heroin 1 day prior to admission and was given a supply of naltrexone by a friend who told him he "wouldn't need heroin" if he ingested it. After ingesting one 50 mg tablet the patient experienced diaphoresis, diarrhea, muscle cramping, and agitation. Mr. B was transported to an emergency department where he was placed in restraints because of disorientation and combativeness. Clonidine, 0.3 mg p.o., was administered with minimal effect. Sedation was achieved with intravenous diazepam. Gastric lavage was performed and the patient was given activated charcoal in sorbitol and admitted. Sedation was maintained with an intravenous infusion of midazolam. On hospital day 3, two TTS-2 clonidine patches were applied, referral was made to a drug treatment center, and Mr. B was discharged.

These cases illustrate two very different rationales for administration of naltrexone. While the dosage of naltrexone was excessive, use in the first case reflected an accurate understanding of the pharmacology of naltrexone and was apparently life saving. Use in the second case represents an irrational application due to ignorance of the properties of naltrexone. This reinforces the need to fully inform patients of the nature of the therapy they receive and to take steps to maximize compliance so that the possibility of diversion is minimized.

Compliance may be increased by administering naltrexone in the presence of a health professional. If naltrexone must be dispensed for home administration, enlisting responsible family members as observers may be useful, but tablet counts remain essential. These measures can reduce the possibility of morbidity associated with diversion and also greatly increase the efficacy of naltrexone as an adjunct in the treatment of opiate dependence.

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Domestic Violence Associated With Anabolic Steroid Abuse

SIR: There is growing concern regarding the psychiatric, behavioral, and medical complications of anabolic steroid use. Illicit use of anabolic steroids for the purpose of enhancing muscular strength and athletic performance has been identified as a substantial public health problem (1, 2). Psychosis and organic manic syndromes, irritability, dysphoria, paranoia, and violent outbursts have been linked to active steroid abuse, while depressive syndromes have been noted during steroid withdrawal (3-5). The significant morbidity of these behaviors in terms of personal injury and property damage, disruption of relationships, and incarcerations for some of these individuals has been noted to be tragic (6, 7). We report a case of child abuse and spouse battery by a man using illicit anabolic steroids.

Mr. A, a 19-year-old college student, began taking steroids on the advice of his football coach. He combined ("stacked") intramuscular testosterone with oral methandrostrenolone over a 4-month period. His size and strength

increased tremendously, and he received positive feedback from the team for his progress and performance. During this steroid use, however, he became increasingly irritable. This caused problems at home, where he was irritable and "rough" with his wife, both physically and sexually, and became increasingly impatient and punitive with their 2-year-old son. Finally, in an effort to discipline the child, he scalded the boy's buttocks with boiling water. The child was temporarily removed from the home, the patient was expelled from the team, thus losing his college scholarship, and his marriage eventually ended in divorce. Upon cessation of steroid use, Mr. A's irritability and violent outbursts resolved within a 2-month period. There was no recurrence at follow-up 18 months later.

This case illustrates the potential for domestic violence and the serious social consequences of behavioral changes secondary to anabolic steroid use. Although complications of steroid use, both medical and psychiatric, are increasingly reported in the literature, the general public's impressions and underground propaganda perpetuate the view that physicians' concerns regarding these complications are exaggerated (2, 4). It has been noted that the scope of this problem is currently underestimated (3). The social consequences are likely underestimated as well, and increased efforts aimed at educating physicians, other health providers, and the general population regarding these risks are warranted.

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Relapse Following Lorazepam Withdrawal

SIR: Clozapine is an antipsychotic drug used to treat schizophrenia in patients nonresponsive to other antipsychotic medications. Some clozapine patients are concurrently taking benzodiazepines. However, recent reports of acute respiratory arrest in such patients have strongly discouraged the simultaneous use of these two agents (1, 2). At our facility these warnings have resulted in a mandate that clozapine patients who are taking benzodiazepines be weaned from them. We report the case of a man who responded well to combined treatment with clozapine and lorazepam. He deteriorated when lorazepam was withdrawn and improved when treatment with lorazepam was resumed.

Mr. A was a 31-year-old white man who had been continuously hospitalized for 13 years with a diagnosis of chronic paranoid schizophrenia. His family reported that he functioned well until 11th grade when he began using marijuana and LSD and drinking beer. His early years in the hospital were characterized by frequent assaultive behavior, prominent auditory hallucinations, and a severe thought disorder. The patient was generally refractory to treatment with multiple standard neuroleptics. Prior to clozapine therapy he was receiving trifluoperazine, 80 mg/day, and lorazepam, 9 mg/day. For 6 years he had been receiving large doses of lorazepam as an adjunctive medication. The lorazepam decreased his hostility and pacing and increased verbal responsivity. Some of the initial improvement diminished over time. Before clozapine treatment was started he spent his days pacing around the ward, appearing disheveled and singing tunes from the 1970s. Approximately once a week he threw furniture and screamed, "The voices, stop the voices." Mr. A rarely spoke to people and when he did speak communications were circumstantial and tangential.

Clozapine treatment of up to 600 mg/day, while the lorazepam dose remained constant at 9 mg/day, brought marked improvement. Mr. A no longer heard voices and was no longer violent or agitated. Instead of avoiding people he sought out conversations with staff and regularly used an honor card to leave the ward. His speech was coherent. He continued to participate minimally in all structured hospital activities. His grooming remained poor. After remaining on this clinical plateau for 6 months, the patient was withdrawn from lorazepam over a 7-month period. During the final months of the withdrawal schedule a gradual deterioration occurred such that by the end of the withdrawal period Mr. A had stopped talking to people, refused to leave the ward, and on one or two occasions reluctantly admitted to hearing voices again. Two months after the lorazepam was discontinued he attacked another patient. Shortly after this incident lorazepam was restarted at 1 mg b.i.d. Within 3 weeks his clinical condition reverted to what it had been before tapering of the lorazepam.

We cannot know with certainty why Mr. A apparently benefited from resuming lorazepam treatment. One explanation is that the lorazepam was treating a lorazepam withdrawal syndrome. If so, there was no abatement of the withdrawal syndrome even 2 months after the lorazepam was stopped and after 7 months of gradual tapering. Another possibility is that, as was the case when Mr. A was on standard neuroleptics, lorazepam was acting as an adjunctive medication, only this time in conjunction with clozapine. A third rationale is that lorazepam had its effect by altering the plasma clozapine level. Little has been written about any of these possibilities. Benzodiazepine-clozapine interactions deserve much more attention.

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Psychotropic Medications and Priapism

SIR: Priapism is a pathologically prolonged erection, usually unrelated to sexual stimulation, that constitutes a urologic emergency. The relationship between priapism and psychotropic medications has been largely disclosed and has been reviewed by Thompson et al. (1). I would like to report a case of priapism that occurred in a patient treated with flupenthixol, a thioxanthene used in Canada as an antipsychotic. A literature search did not reveal any previously reported cases of flupenthixol-induced priapism.

Mr. A was a 26-year-old patient admitted for a psychotic relapse. His past psychiatric history revealed 10 admissions with a diagnosis of paranoid schizophrenia and mild mental retardation. During previous admissions he responded well to antipsychotic medication, either haloperidol or flupenthixol (up to 9 mg/day), without any major side effects. His medical history was clinically insignificant. He had stopped his medication (flupenthixol and benztropine) a few weeks before his relapse.

Flupenthixol was started with benztropine upon admission and was increased gradually to 18 mg/day, without major side effects. This led to a remission of the psychotic symptoms and the disruptive behavior. He was maintained in the hospital on 18 mg/day of flupenthixol taken orally and benztropine, 2 mg b.i.d., for a month when he developed a painful and sustained erection that required emergency surgery. The dosage of flupenthixol was reduced to 12 mg/day along with continuation of oral benztropine, 2 mg b.i.d., without recurrence of priapism, and Mr. A was able to achieve normal erections.

Priapism has been reported with various psychotropic agents (1-3). The most frequently proposed mechanism for psychotropic medication-induced priapism is an increased parasympathetic tone in relation to sympathetic tone through a direct α blockade that leads to an inhibition of the detumescence process (1). Thioxanthenes, such as flupenthixol and thiothixene, seem to have high α -adrenergic blocking properties. The patient was also taking benztropine. Anticholinergic effects on sexual function have been mainly reported as delayed or retrograde ejaculation and difficulty in maintaining erection (4). Much controversy exists about effects of antiparkinson agents on antipsychotic blood levels (4). Since most of the cases reported implicate antipsychotics with high α -adrenergic blocking affinity, flupenthixol was considered the offending agent.

This case illustrates the reported absence of relationship between the length of treatment and the emergence of priapism (1), as our patient was treated with flupenthixol for prolonged periods in the past without any signs of priapism. In this case, priapism occurred at a dose of 18 mg/day, which was the highest dose the patient ever received. A reduction of the dose to 12 mg/day after the onset of priapism did not lead to a recurrence of this side effect, raising the possibility that it could have been a dose-related effect, contrary to other reports (1).

As more cases of psychotropic drug-induced priapism are reported, it seems that particular attention should be paid to this potential side effect when prescribing potent α blocking agents.

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Sleep Deprivation in an Elderly Man With Parkinson's Disease

SIR: We would like to report an 81-year-old man with depression and Parkinson's disease who was successfully treated with sleep deprivation. Cummings (1) reported an approximate 40% comorbidity of depression and Parkinson's disease. Although the pathophysiology of Parkinson's disease and depression is not well understood, evidence suggests involvement of noradrenergic and dopamine pathways within mesolimbic structures (1).

Mr. A was hospitalized with a 13-year history of concomitant Parkinson's disease and "intolerable" depression which included diurnal variation, anhedonia, poor appetite, diminished concentration, and sleep disturbance. His symptoms of Parkinson's disease were being maintained on carbidopa-L-dopa. However, his depressive symptoms were unresponsive to therapeutic trials of phenelzine, tranylcypromine, fluoxetine, nortriptyline, bupropion, lithium, methylphenidate, and 19 sessions of ECT. Due to the refractory nature of his depression, sleep deprivation was initiated.

The patient was put to bed at 8:00 p.m. and awakened at 1:00 a.m. He remained awake in a well-lit and ventilated room until returning to sleep at 11:00 p.m. the following night. Hamilton Rating Scale for Depression (2) scores were recorded at 5:00 p.m. prior to the initiation of sleep deprivation and at 8:00 a.m. and 5:00 p.m. following the onset of treatment. The scores were 27, 7, and 15, respectively. During the morning hours of the sleep deprivation trial Mr. A appeared with bright affect, an improved mood and appetite, and a decrease in speech latency and psychomotor retardation. He reported that he was feeling the best he had felt since 1979. However, by 5:00 p.m. he appeared to relapse, evidencing increased agitation, anxiety, and depressed mood. He was started on a regimen of lithium, 300 mg/day. Throughout the following week he demonstrated a steady decline beyond his original baseline level. On the seventh day of lithium treatment, sleep deprivation was readministered. His Hamilton depression scale score was 27 at 5:00 p.m. prior to the second sleep deprivation trial. Following the sleep deprivation intervention he scored a 5 on the Hamilton depression rating scale (at 5:00 p.m.). He was discharged 2 days later with an increase in affective range, improved concentration and attention, and amelioration of his depressed mood and feelings of hopelessness. His wife contacted the staff 4 weeks after discharge to report that he maintained his nondepressed state and that she had "her old husband back."

It would appear that sleep deprivation reduced the depressive symptoms of this patient with Parkinson's disease, or at the very least potentiated the effects of lithium on our patient. Although little is known about the effects of sleep deprivation on depression, some have suggested that the effects of sleep deprivation

are dependent upon dopamine and norepinephrine (3). Thus, the therapeutic effects of sleep deprivation in a patient with Parkinson's disease may be attributed to enhanced activity of dopamine in the dopamine-depleted ventral tegmental region.

Sleep deprivation is a safe and well-tolerated procedure which may prove beneficial in a subpopulation of depressed patients who have failed to respond to more traditional therapeutic approaches.

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Failed Suicide or Successful Male Genital Self-Amputation?

SIR: Genital self-inflicted wounds are generally categorized as self-mutilating behavior or partial suicides (1) but not considered failed suicides (2). "Klingsor syndrome" reflects the perception that such behavior is an act of emasculation (3). Schweitzer reported a Chinese man who made the "ultimate sacrifice *intending* [emphasis added] to end his life" (4). We recently managed cases of three Chinese men with isolated penile amputations, where each patient's expectation had been death. Cultural beliefs may explain perceptual differences.

Mr. A, 32 years old, was brought in by a coworker after being found to have amputated his penis with a kitchen knife. His girlfriend had recently left him. He had chronic auditory hallucinations from various deities. That morning one deity voiced, "A big hero like you should not fear death; you will soon be reincarnated." Mr. A had a history of pentazocine abuse and schizophrenia but had never received regular treatment. After successful recovery from replantation he discharged himself against medical advice. Five months later, he reamputated his penis and completed suicide by jumping from the 7th floor of the receiving hospital.

Mr. B, 25 years old, was brought in by his parents, having severed his penis with a saw. He believed this would lead to excessive bleeding and death. He felt his continuing amphetamine abuse had brought great shame to his family. Over the past year Mr. B heard persecutory voices including the voice of Buddha criticizing his sexual lust. Two weeks after his operation the patient removed his catheter and attempted to jump from the 10th floor window. Neuroleptics were successfully increased, then discontinued by the psychiatrist 2 months later. Mr. B remained both amphetamine and delusion free while working happily in the family business.

Mr. C, 28 years old, amputated his penis with scissors. He repeatedly screamed that he had cut off his penis ("Ming Gon") and was puzzled and angry at still being alive. He was

acutely psychotic, with 2 months of auditory hallucinations after a breakup with his girlfriend. Since his successful re-plantation, neuroleptics have controlled his schizophreniform disorder.

Many Chinese continue to believe that mental illness is incurable and brings great shame to the family. This often brings overt or covert pressure to the patient to commit the final act. In a review by Greilsheimer and Groves of 53 cases of genital self-mutilation, five were considered suicidal, with one confirmed suicide (1). Schweitzer identified 20 further cases, suggesting that the suicide risk is high due to the extent of psychopathology (4). Our experience with these three cases leads us to wonder whether Schweitzer's Chinese patient didn't make the "ultimate sacrifice" *expecting* to end his life. In Chinese, one term for penis is "Ming Gon" meaning "life root" or "life source." In each of our cases, the patient believed that by severing this life source, death was inevitable, and each expressed surprise and dismay at finding himself still alive. We recommend approaching Chinese genital self-mutilators as failed suicides with genital injuries rather than self-mutilators who may be suicide risks.

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Driving and Psychiatric Illness

SIR: The letter by Leo E. Hollister, M.D. (1), adds weight to the demand for research into the effect of psychiatric illness on driving ability (2). While psychiatrists and other physicians are often involved in decision making about ability to drive, the absence of guidelines reflects the difficulty in relating psychiatric or psychological assessments with driving ability (3, 4). Variations in the requirements of licensing authorities underline the lack of consensus on the issue.

One helpful suggestion is to use a hierarchical approach for decision making (5), a strategy already proposed for brain injury (6) and illness in the elderly (7). This divides the driving task into three levels of risk taking: strategic, tactical, and operational. Strategic performance is the driver's choice of level of risk. This includes the planning of choice of route, time of day, or even the decision not to drive and to take public transportation. Tactical decisions are those which take a risk, e.g., decisions on overtaking, going through amber lights, or signalling in good time. Operational performance is the reaction to risk, i.e., the response to specific traffic situations, such as speed control, braking, and signalling. Driving a car requires organization of action at all three levels.

Assessment up to now has tended to dwell on deficiencies on the operational level, i.e., whether an illness affects the subject's appreciation of distracting stimuli or the reaction time to a hazardous situation. This emphasis is misguided: reaction time (a measure that is an integral part of operational

tasks) is shortest in the 15-25 age group, the group with the highest accident rate. Older drivers are known to use strategic and tactical measures to avoid delay, stress, and risk by driving less at night and during bad weather and by avoiding rush hours and unfamiliar routes. It is very likely that decisions at a strategic and a tactical level are more important than operational factors in causing accidents, particularly in patients with psychiatric and neurological diseases. This framework may aid in decision making for physicians in the absence of clearer guidelines.

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Dr. Hollister Replies

SIR: Dr. O'Neill offers a systematic method for appraisal of driving impairment that has been useful in elderly and mildly demented drivers. It will be interesting to see whether such an assessment is applicable to patients with major mental disorders such as schizophrenia, mania, or depression. The course of those illnesses is much more variable than in the case of a patient with incipient Alzheimer's disease. Patients usually apply for drivers licenses when they are in at least partial remission. But how might testing at that time predict the situation several months hence? If the assessment described could be made applicable to other types of psychiatric patients and were it to be proved valid by subsequent experience, it might help solve the difficult problem of predicting driver safety for individual patients. Let's hope to hear more when such studies have been done.

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Update on the Dementia Spectrum of Depression

SIR: The recent article by V. Olga Emery, Ph.D., and Thomas E. Oxman, M.D. (1), brings refreshing novelty to the debate on the relationships between depression and dementia. It relies on an extensive review of the relevant literature and transmits a complete picture of the present state of research in all areas of the field. There are several points regarding the conclusions drawn from the reviewed data to which I would like to add.

I agree with the authors' opinion that the cognitive deficits seen in some depressive syndromes in old age are "real, not pseudo or simulated" and found judicious their comment stating that data about major depression cannot be extrapolated to depressive dementia. The observation cited from Lipowski (2) concerning the essentially pragmatic nature of diagnostic categories seems quite apropos.

Given these considerations, I feel the authors should have laid more stress on the importance of developing strict operational definitions. From a clinical standpoint, the proposed continuity concept, where dementia and depression stand at both ends of a spectrum, is interesting. By leading the clinician away from a dichotomous and rather reductionist viewpoint, such an approach should favor a more adequate, complete, and personalized treatment for the patient.

From a research perspective, however, while the continuity conception stimulates a certain reorganization of current knowledge, it remains rather "unpractical" for instrumental purposes. The operationalization of such a concept is borne with problems and requires the definition of several typological groups. In fact, while they discuss with justness the imperfect quality of our present empirical classification systems, the authors paradoxically appear to corroborate the dichotomous conception by presenting dementia and depression as different disease processes with etiologically distinct, albeit frequently concurrent, manifestations. It would seem more realistic for the moment to concentrate on refining and validating our general diagnostic categories (e.g., depression) for elderly populations and, specifically, for subjects presenting with cognitive deficits; the operational definition of dementia itself still allows for greater refinement.

The approach best fitted to attain such an objective remains the clinical characterization (descriptive, phenomenological, symptomatic) of demented patients with depressive features, along with a multidisciplinary contribution (neuropsychology, neuropathology, etc.). It should rely first on the study of clear-cut, specific symptoms (3) rather than empirical, syndromic definitions of depression with no sound basis for these populations. The main clinical objective (as stated by the authors) would be to delineate which clinical constellations are liable to respond to treatment. The ultimate theoretical aim is to provide evidence toward a better comprehension of this complicated relationship; only a well-documented understanding will enlighten these intricacies.

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SIR: In their otherwise superb review on the dementia spectrum of depression Drs. Emery and Oxman refer both to the "consistent" low rate (i.e., 1%) of major depression in subjects older than 65 and to the (related) inverse relationship between the prevalence of major depression and age. The authors did not cite any supporting literature, but their statements seem to be based on the results of the widely quoted

Epidemiologic Catchment Area (ECA) study that reported a prevalence of major depression of 1% in 5,000 persons aged 65 and over, compared to 2.3% and 3.4% in persons 45-64 and 18-44, respectively (1, 2). Considering the unequivocal associations between physical illness or suicide and both major depression and age, the results of the ECA study remain puzzling.

Snowdown (3) recently reviewed potential reasons for the discrepancy between the ECA study and several other recent studies of the prevalence of depression in late-life conducted in the United States, Canada, Europe, or Australia. Of relevance to the dementia spectrum of depression, a significant proportion of elderly with major depression reside in institutions (4, 5), but the ECA results published so far pertain only to persons living in the community. Furthermore, following the methods of the ECA study, Blazer et al. (6) found again that less than 1% of community-dwelling elders were classified as suffering from major depression. However, an additional 7% were classified as suffering from either "symptomatic depression," "mixed depression/anxiety," or dysthymia. Interestingly, the proportion of patients presenting with some cognitive impairment was similarly high in these four groups (70%, 44%, 25%, and 48%, respectively; calculated $\chi^2=5.23$, $p>0.10$).

In conclusion, we suggest that the relationship between the prevalence of major depression and age remains an open question and that, when analyzing the interaction between depression and dementia, it may be misleading to assume that the prevalence of major depression in late life is only 1%.

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Dr. Emery and Dr. Oxman Reply

SIR: We thank Dr. Paquette and Drs. Mulsant and Reynolds for their kind comments regarding our article. Dr. Paquette emphasizes that while the spectrum approach to the relationship between depression and dementia has both theoretical and clinical utility, it is "unpractical for instrumental purposes." Specifically, Dr. Paquette is concerned about the numerous problems that would be involved in the operationalization of the continuous relation between depression and dementia. We concur with Dr. Paquette that stricter opera-

tional definitions are needed for both depression and dementia, and for that matter, for all constructs, i.e., concepts and their operationalization (1) of mental disorders. However, we maintain that fundamental problems of operational validity and reliability increase rather than decrease the need for concepts that reflect empirical reality. An operational definition can be no more accurate than the concept it purports to indicate and operationalize. Practically speaking, it seems we should retain depression and dementia as separate nosological categories, as they now exist in DSM-III-R. The continuous relation between the diagnostic categories in DSM-IV might be operationalized by the following: 1) adding to mood disorders additional criteria and/or fifth-digit code numbers to designate the existence and severity of cognitive impairment; 2) adding to primary degenerative dementia additional criteria and/or fifth-digit code numbers to designate the existence and severity of depression; and 3) not using diagnostic labels based on the false dichotomization of organic versus nonorganic.

The concern of Drs. Mulsant and Reynolds regarding depressive conditions in the elderly is an excellent example of both the necessity for and the difficulty in making sense of the continuous variability in depressive symptoms among the elderly. Although we agree with their concern about the uncertain rates and meaning of possibly different types of depression in the elderly, their conclusion regarding rates of major depressive disorder and dementia is less clear.

We were indeed referring to the consistently low rates of major depression in subjects 65 and older as determined primarily across the sites of the ECA study (Drs. Mulsant and Reynolds' reference 2). There are other rigorous comparative studies, such as the Stirling County study (2), which also suggest that "severe" depressive disorders—equivalent to major depression—are less prevalent among the elderly.

Drs. Mulsant and Reynolds make the observation that the "unequivocal associations between physical illness or suicide and both major depression and age [make] the results of the ECA study . . . puzzling." Physical illness and suicide are certainly more prevalent among the elderly, but it does not necessarily follow that major depression is or would be more prevalent. On the one hand, ECA investigators themselves (Drs. Mulsant and Reynolds' reference 1) have suggested several plausible reasons why major depression may actually be higher in later life than reported in the ECA study. These reasons include historical increase among the young, less willingness to report symptoms, disorder-associated mortality, and difficulty in recall. On the other hand, Neugarten (4), Erikson et al. (5), and Barton (6) have pointed out how the elderly are better able to tolerate losses because of their expectation of loss, as well as their longer life experience, leading to greater knowledge. That this expectation and experience make the elderly less prone to major depression is further suggested by the lower rates of major depression (as opposed to adjustment disorder) in older, hospitalized, medically ill veterans compared to younger hospitalized veterans (7).

With respect to the relationship of depression to degenerative dementia, the "cognitive impairment" associated with non-DSM-III subtypes in the ECA study of Blazer et al. (Drs. Mulsant and Reynolds' reference 6) should be considered in the context of the low educational level of that sample, of the acknowledged educational bias of the Mini-Mental State exam, which was used to define "cognitive impairment" in the ECA study (4), and of the finding that the only significant group difference reported by Blazer et al. was for "mild cognitive impairment," not "severe." Given this context, it is perhaps most important to realize that comparing Mini-Mental State examination scores is analogous to comparing depres-

sion rating scale scores, not depressive (nor dementia) disorders. Both of these comparisons are related to a problem raised by Dr. Paquette, that is, of operationalizing clinical categories from limited continuous information. What is missing is consideration of another continuum, that of functional impairment (8).

The evidence referred to by Dr. Mulsant and Dr. Reynolds is strongly suggestive of an open question regarding the meaning of mild depressive symptoms in the elderly—do these symptoms comprise non-DSM-III-R disorders that require or would respond to treatment? Whether these nonmajor depressive disorder syndromes will have any relationship to degenerative dementia is also an interesting question. However, based on the available research we do not think it misleading to assume that the prevalence of late-life major depression—the affective disorder most clearly associated with dementia—in the community is far less than the prevalence in earlier life, nor is it misleading that the rate of major depression decreases in later life while the prevalence of degenerative dementia increases.

The higher rate of major depressive disorder in nursing homes should be put in the context of the much higher rate of dementia disorders found in nursing homes. The ratio of the prevalence of dementia to the prevalence of major depression in this setting is as much as 10 to 1 (Drs. Mulsant and Reynolds' reference 4), a ratio that may be similar to that found in community studies. As Dr. Paquette points out, we especially need improved clinical characterization of demented patients with depressive features—including longitudinal history—to better make sense of what these ratios can tell us about the relationship of depression and dementia.

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Pain in Depression and Parkinson's Disease

SIR: Jeffrey L. Cummings, M.D. (1), has provided a useful review of the association between depression and Parkinson's disease. However, he omitted any discussion of an issue of vital importance: the relationship between these disorders and pain. Depression is frequently found in patients suffering from chronic pain (2), and pain is a common symptom of Parkinson's disease (3).

Unfortunately, by excluding pain from his review, Dr. Cummings inadvertently demonstrates psychiatry's failure to recognize the importance of this subject and perpetuates the myth that psychiatrists should only be interested in pain that is considered somatoform or psychogenic in nature and neglect that in which organic factors appear to have a major etiologic role. This may result in needless suffering because, for certain Parkinson's disease patients, the correct management of their pain may be a key factor in the resolution of their depression (4). Adoption of the proposal for creation of the diagnostic category of pain disorder in DSM-IV (5, 6) may help psychiatrists recognize the significance of pain and take the lead in its treatment.

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Dr. Cummings Replies

SIR: Dr. King emphasizes an important point. Pain can be an important aspect of the neuropsychiatry of Parkinson's disease. The reported frequency of pain in Parkinson's disease varies considerably from involvement of as few as 12% of patients in some studies (1) to as many as 46% in others (2). Goetz et al. (2) provided a useful classification of painful syndromes in Parkinson's disease, dividing them into painful cramps of the neck and spine, painful dystonia, radicular or neuritic pains, joint pains, and the diffuse discomfort of akathisia. Although depression was no more common among patients with and without pain (63% and 56%, respectively), depression rating scale scores were significantly higher in the patients experiencing pain, suggesting that depression was more severe in those with painful disorders. The observation that pain is most intense when patients with the on-off phenomenon are in the off state indicates that L-dopa may participate in central analgesic activities (3). One caution in regard to the potential role of pain in depression in Parkinson's disease concerns the high frequency of pain in elderly individuals without neurologic disease. Reid (4) reported high rates of pain in a control population, as well as among patients with Parkinson's disease.

Pain assessment should be included in the evaluation of patients with Parkinson's disease. Investigation of pain in Parkinson's disease may shed additional light on the role of L-dopa in neuropsychiatric syndromes.

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Diagnostic Criteria for Multiple Personality Disorder

SIR: Kenneth Nakdimen, M.D. (1), cited Ross et al.'s 5% frequency for multiple personality disorder (2) in psychiatric inpatients and the implied underdiagnosis of the condition as evidence that the DSM-III-R criteria lack sensitivity. He proposed changes to increase this sensitivity, but those changes would be of little utility as Ross et al.'s estimates are based on DSM-III-R criteria.

The problem of clinicians not considering the dissociative disorders will not likely be solved by Dr. Nakdimen's proposed criteria A, B, and C. If changes in criteria are to be aimed at this problem, a more effective change would be to make the presence of multiple personality disorder an exclusion criterion for conditions often confused with it (schizophrenia, major depression with psychotic features, etc.).

Dr. Nakdimen's proposal to add amnesia as a criterion does add some rigor to the diagnosis and in that vein is almost identical to that proposed already for DSM-IV (3).

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Dr. Nakdimen Replies

SIR: The current diagnostic criteria for multiple personality disorder take it for granted that the alternate personalities will be immediately recognizable as such. But clinical reality is that in most cases all you will be aware of is a puzzling changeability or incongruity of the person (my criterion A) because the usual prediagnostic status of most alternate personalities is either "out" but incognito (deceptively answering to the person's regular name) or covert (pulling strings, but rarely coming "out"). The current criteria, though useful in research (as a standard definition to ratify a diagnosis that has been arrived at by other means), are simply not descriptive of the usual presentation and so fail their clinical role.

The plan of the DSM-IV Task Force to add amnesia to the DSM-III-R criteria will be to little avail clinically, because persons with multiple personality tend to cover up and avoid the issue. In most cases, if you don't ask the kind of questions listed in my criterion B, you'll never know that the patient has been having amnesia.

My criterion C differentiates multiple personality from

moods and ego states, neither of which, as I use the terms, have their own separate identities and memory banks. I also try to help rule out malingering and iatrogenic responses by requiring that the multiplicity antedate legal problems and psychiatric evaluations.

I agree with Dr. Wetsman that multiple personality disorder should be included in the differential diagnosis of adult disorders with which it may be confused (1). It is also common sense that a disorder with a childhood onset should be included in the differential diagnosis of children's disorders and even have its own listing in that section of DSM.

With all these measures, perhaps we can do something about the fact that the average multiple personality patient today has had to suffer the condition for literally a quarter of a century before being correctly diagnosed, and that most such patients never get diagnosed.

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Treatment of Stuttering With Phenelzine

SIR: The recent article by John Paul Brady, M.D. (1), is a useful review of the pharmacotherapy of stuttering. However, it neglects the possible use of monoamine oxidase inhibitors (MAOIs) for the treatment of stuttering complicated by social or performance anxiety. MAOIs are useful in the treatment of social phobia (2). Social and performance anxiety frequently develop as a consequence of stuttering (3) and would meet criteria for social phobia except that DSM-III-R, without empirical basis, excludes social anxiety secondary to axis III conditions from the diagnosis of social phobia. We report a case of stuttering complicated by social anxiety ameliorated by phenelzine.

Mr. A, a 24-year-old Caucasian single man, had a family history of stuttering and stuttered since age 5. Social anxiety around speaking worsened after puberty. In his teens, speaking in public was preceded by severe anticipatory anxiety with sweating, shaking, and blushing. Anxiety over speaking precipitated college attrition and ensuing demoralization with reclusive social withdrawal for 3 months but no neurovegetative changes. Treatment with imipramine, alprazolam, and speech therapy was ineffective.

Upon evaluation, Mr. A met DSM-III-R criteria for social phobia except for the secondary nature of his social anxiety. Speech was often inaudible and eye contact poor. Stuttering was characterized by repetition and prolongation of first syllables, mainly on consonants *d* or *p* on words located randomly throughout a sentence. Self-perception of stuttering may have exceeded actual difficulty.

Given the lack of data differentiating social anxiety secondary to stuttering from social phobia, we treated Mr. A with phenelzine. He was started on a dose of 15 mg/day, and increased by 15 mg/day every 7-10 days. After 4 weeks on phenelzine, 60 mg/day, he reported more confidence in expressing himself. He no longer feared getting "stuck" on a word, felt less self-consciousness and anticipatory anxiety, noted no hyperautonomic symptoms, and became more ex-

troverted. He spoke more audibly and spontaneously with considerably less stutter.

After 6 weeks on phenelzine, 75 mg/day for 10 days, he ingested some vodka, then became floridly manic, with overspending, rapid and pressured speech, and poor social judgment. In this manic state he no longer stuttered. Psychiatric hospitalization was necessary; phenelzine was discontinued and lithium begun. Four days later, stuttering returned, though to a milder degree.

Given the lack of bipolar history in the patient or family, we believe the patient had a toxic manic reaction to phenelzine. The temporary improvement in social anxiety and stuttering with phenelzine suggests the need for reconsidering the relationship between social anxiety secondary to stuttering and social phobia and possible treatment of stuttering plus secondary social anxiety with MAOIs.

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Dr. Brady Replies

SIR: Persons who stutter are seldom indifferent to the presence of listeners. Indeed, most stutterers, even very severe ones, are usually perfectly fluent when speaking out loud when no one else is present. The amount of anticipatory anxiety generated in a particular speaking situation differs with different stutterers. In some, performance anxiety is so intense in ordinary social situations that a social phobia results, as probably was the case with the patient described by Dr. Oberlander and associates.

On entering a situation in which he or she is expected to speak, the stutterer experiences anticipatory anxiety, which produces additional tension in the muscles used in phonation. This ensures further disruption in the flow of speech, thus confirming the patient's fears. Further anxiety develops when the speaker later enters similar speaking situations. Thus, the stutterer's dysfluency is in a kind of positive feedback loop; stuttering begets more intense anticipatory anxiety, which in turn aggravates the speech problem.

Anxiolytic agents have some use in the early treatment of stuttering by halting or reversing this escalation of anxiety and associated dysfluency. MAO inhibitors may be useful in patients who have developed social phobias in response to their intense embarrassment in speaking situations. However, the use of either class of drug is not likely to be sufficient treatment for severe, adult stutterers. My review describes drugs that appear to promote fluency not by an anxiolytic action but probably by a more direct effect on the desynchronization of vocal motor functions that characterizes this perplexing disorder. However, the most promising approach to severe stut-

tering in adults is a multicomponent program that combines pharmacotherapy with individual or group psychotherapy. The latter best includes elements of supportive psychotherapy, cognitive restructuring, and behavioral procedures directed at reshaping fluency (1).

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Statistical Problems With Small Sample Size

SIR: Manuscripts published in the Clinical and Research Reports section of the *Journal* often present suggestive findings from pilot studies. However, because these studies usually employ a relatively small number of subjects, e.g., N fewer than 50, statistical power to detect a true effect is likely to be low: that is, there is little a priori chance of rejecting the null hypothesis (1). Statistical power is often especially low when costly medical imaging technologies such as positron emission tomography or magnetic resonance imaging (MRI) make it prohibitively expensive to study large numbers of subjects. Consequently, findings presented in these reports may be erroneous, particularly in the case of negative results (2).

As an example, H. Jordan Garber, M.D., and Edward R. Ritvo, M.D. (3), recently reported the use of MRI to compare several posterior brain areas between 12 autistic patients and 12 matched normal controls to assess, among other areas, the size of the fourth ventricle. These authors reported nonsignificant differences in volume and area of the fourth ventricle between the groups by t test ($p < 0.11$ and $p < 0.10$, respectively), and since p was greater than the conventional 0.05, concluded that the results did not confirm earlier findings of abnormal fourth ventricles in autism. The mean volumes of the fourth ventricle were 1.11 cm³ and 0.99 cm³ for the autistic and control groups, respectively; with a difference of 0.12 cm³ and average SD of 0.18, the effect size for the t test is calculated as 0.69 SD units, which falls between Cohen's (1) conventions of 0.50 SD and 0.80 SD as "medium" and "large" effect sizes. Given all the elements required to calculate power— p value (0.05), effect size (0.69 SD), and number per group (12)—reference to Cohen's (1) tables gives power=0.37. That is, even if the null hypothesis is in fact false, this study had only a 37% chance of rejecting it by detecting a medium-to-large effect size, calling into question this "negative" finding.

Although Dr. Garber and Dr. Ritvo (3) appropriately pointed out that nonsignificant correlations between age and pontine area in their autistic subjects may have been due to type II error (e.g., low power), they failed to note that their major finding also may have been due to low statistical power.

We suggest that for primary analyses that yield negative results with a small number of subjects, authors report the effect size of the treatment (1) and the power of the statistical test, in addition to p values, to permit the reader to interpret a negative result appropriately.

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LEE BAER, PH.D.
DAVID K. AHERN, PH.D.
Charlestown, Mass.

Dr. Garber Replies

SIR: Drs. Baer and Ahern exemplify the statistical problems associated with small sample size with our MRI study of adult autistic patients and comment that our "major finding . . . may have been due to low statistical power," referring to volume measures of the fourth ventricle. I have three points in response.

First, our recent MRI study was done because our earlier MRI study of young autistic patients (1, 2) did not replicate the findings of Courchesne et al. (3), but comparisons were limited because our first group was younger than the group studied by Courchesne et al. Also, midsagittal area of the fourth ventricle, which Gaffney (4) had found to be larger in autistic patients, was not abnormal in our initial MRI study of young patients with autism (1). The major statistical finding of our recent study was that neither of these measures differed between adult autistic and normal groups. The fourth ventricle volume comparison was included as a secondary level of data analysis.

Second, sample size calculations based on the data of Courchesne et al. (3) and Gaffney (4) indicate that 11 subjects per group yields an 80% chance of detecting a difference for either area of vermis lobules VI and VII or fourth ventricle area (with the usual assumptions: type I error rate of 0.05, type II error rate of 0.20); we studied 12 in each group. Fourth ventricle volumes from our initial MRI study of young autistic subjects and normal subjects (1) demonstrated an effect size of only 0.25 SD; sample size calculation predicts that more than 240 autistic and more than 240 normal subjects would be needed to have an 80% chance of finding that between-groups difference as statistically significant. We did not study larger samples because the effect sizes for Courchesne et al. (0.81 SD) and Gaffney (1.21 SD) were large compared to our fourth ventricle volume data.

Drs. Baer and Ahern used the fourth ventricle volume data from our recent study (effect size of 0.69 SD) to conclude that we had only a 37% chance of detecting the observed difference as significant. Unfortunately, results from our study were not available for us to use in advance for considering sample size; rather, calculations made from published data show that the sample size used was adequate for the specific task.

Lastly, the most important test of meaning for a statistical finding is the clinical significance. In our study, the 12% difference in mean values for fourth ventricle volume was not a clinically valuable result, since this could not be detected on an individual basis for diagnosis and did not clearly implicate posterior fossa development in autism. The most significant results from our initial and follow-up MRI studies of autism are that neither vermian hypoplasia nor gross abnormalities are associated with autism. These are important negative findings because they may save patients and families the expense, discomfort, and false hopes of an MRI procedure, which would be unproductive unless a high index of suspicion exists for an abnormality likely to be detected by MRI. The comments of Drs. Baer and Ahern may be misleading if taken to mean that even statistically weak and clinically insignificant observations must be exhaustively disproven.

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H. JORDAN GARBER, M.D.
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Seasonal Patterns of Bulimia Nervosa

SIR: The study by Arthur Blouin, Ph.D., and colleagues (1) suggests seasonal patterns in the occurrence of bulimia. Since in bulimia the menstrual cycle is often affected, a comparison of the seasonality of both phenomena seems warranted. Seasonality of menarche has been extensively reported. Recently I tested this agreement with the time series of menarche based on the input of at least 1,000 Caucasian school girls (2). Seven such series of subjects were found in the literature. Kendall coefficient of concordance appeared to be significant ($p=0.01$). The similar seasonal trend of the seven series allowed the addition of the monthly numbers of each of the series, after reducing their totals to 1,000 subjects. A correction for unequal length of months was also made. Beginning with January these numbers are, 970, 485, 456, 519, 483, 545, 542, 600, 596, 547, 631, and 626.

Comparing the monthly variations of menarche and binge eating reveals several interesting features. Both the peaks of menarche prevalence and mean binges per week occur in January (data on bulimia deduced from figure 2 [1]). Both peaks are followed by a sharp decline during February. The quarter with the highest numerical values for both is November-January. The average monthly increase during these 3 months, as compared to the average of the other 9 months, is 30%. These similarities may point to the possibility that the biological factor(s) leading to enhanced expression of menarche may also contribute to exaggerated binge eating.

With regard to purging, the situation is less clear: here the trough occurs in January. It might be that purging reacts earlier to the influence of dark hours or to another factor contributing to the proxy variable, so that the data of purging must be shifted forward by 1 month. Another indication of enhanced reactivity to environmental factors of purging is that the quarter with the highest mean purges per week precedes the quarter of binge eating by 1 month: October-December versus November-January. Again in this quarter there is a 30% increase of purges, as compared to the mean of the other quarters.

The finding of similar temporal patterns in the symptoms of bulimia and developing reproductive ability is concordant with the tendency of bulimia to start in women in late adolescence or early adulthood. The lack of such an association may be one reason explaining why the disorder is uncommon in men.

The figure of 30%, which appeared earlier, may indicate heterogeneity in bulimia similar to the heterogeneity proposed in endogenous depression (3). This is consistent with my working hypothesis, which ascribes to a subset comprising one-third of the (Caucasian) population specific genetic characteristics that control endocrinological, metabolic, and rhythmic events (4).

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PHILIP COHEN, D.M.V.
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Is Childhood Sexual Abuse a Risk Factor for Bulimia?

SIR: While providing an excellent compendium of studies relevant to childhood sexual abuse and bulimia, Harrison G. Pope, Jr., M.D., and James I. Hudson, M.D. (1), have ignored two issues, one definitional and the other clinical.

Bulimia is a behavior-based diagnosis. As such, it ignores certain differentiations which may have critical importance in etiological research. These differentiations include that the type of purging (vomiting, laxative use, or both); the type of onset (individual versus in a social setting such as during hospitalization); and premorbid level of obesity.

More importantly, the authors ignored the nature of repression. Although they do refer to "amnesia or 'screen memories' for traumatic experiences of sexual abuse," they concluded that this would be true of the general population as well as those with bulimia and go on to suggest that because of therapy patients with bulimia might actually have better recall of abuse than control subjects. First, it is frequently a matter of many years of therapy for any patient to be able to recall sexual abuse. Second, if we consider bulimia a hysterical conversion, we must recognize that such a dynamic may greatly reduce recall of the earlier trauma. Indeed, considering bulimia nervosa as a conversion symptom raises questions as to how some patients with bulimia recall sexual abuse at all.

Of course, considering bulimia nervosa to be an hysterical conversion does lead to a double bind. The recall of the trauma becomes evidence of the trauma and the nonrecall of the trauma becomes evidence of the trauma. How do we escape from such a conundrum? One possibility would be the use of projective testing. While psychometrics would not give evidence of early abuse, it can give evidence of repression, hysterical reaction, and attitudes toward sexual content. Obviously, the appropriate comparison group would be persons with other eating disorders.

It is important that we not allow present-day preoccupation with behavior and symptom control to lead to institutionalized repression of our knowledge of the unconscious.

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KENNETH A. WEENE, PH.D.
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Dr. Pope and Dr. Hudson Reply

SIR: Dr. Weene first argues that the behavior-based diagnosis of bulimia may ignore "differentiations that may have critical

importance in etiological research." However, we are unaware of any evidence demonstrating clear differences in the etiology of subtypes of bulimia characterized by different types of purging, types of onset, or premorbid obesity. Even if these types of bulimia were differentiable, it would be mathematically unlikely that one subtype could be associated with increased childhood sexual abuse, but that the rate of childhood sexual abuse in bulimia as a whole would still remain equal to or less than that reported in the general population—as found in our review.

Dr. Weene then suggests that we ignore the "nature of repression." But here we must respectfully challenge him (or anyone else) to provide methodologically sound evidence that repression actually exists. As Holmes (1) has pointed out, more than 60 years of psychological research have failed to produce a clear demonstration of repression. And even if we assume, for argument's sake, that repression really does occur outside of Hollywood movies, what data demonstrate that bulimia is "a hysterical conversion"? Indeed, if bulimia were hysterical, why does it respond to antidepressant drugs but not to placebo in more than 12 double-blind studies (2)?

In conclusion, we share Dr. Weene's concern that one should not neglect "our knowledge of the unconscious." But that knowledge will be dishonestly served if it is based on speculation in the absence of legitimate evidence.

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HARRISON G. POPE, JR., M.D.
JAMES I. HUDSON, M.D.
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Alprazolam in the Emergency Treatment of Schizophrenia

SIR: I read with great interest the article by James G. Barbee, M.D., and colleagues (1), but I would like to comment that in a study designed to investigate the efficacy of treatment for schizophrenic patients in acute psychotic relapse, it is curious that haloperidol levels were under the lower limit proposed by Van Putten and et al. (2).

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JEAN-CLAUDE MONFORT, M.D.
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Dr. Barbee Replies

SIR: In regard to the issue of the haloperidol drug levels we reported, Dr. Monfort notes that it is "curious" that the blood

levels reported in our study were less than those proposed by Van Putten et al. (Dr. Monfort's reference 2) as a therapeutic window for haloperidol. This discrepancy is likely due to basic pharmacokinetic principles and differences in the design of the two studies. The drug levels reported by Van Putten et al. were based upon weekly blood samples drawn over a 4-week period, during which the patients received fixed dosages of haloperidol. In our study the dosage of the drug was determined by the outcome parameters of the protocol and varied widely among individuals. In addition, the drug levels obtained within our study were drawn at 72 hours or less. The elimination half-life of haloperidol varies widely among individuals. When haloperidol is given orally, reported mean values of its elimination half-life have ranged from 14 to 37 hours (1). Under conditions of repetitive dosing, assuming that the plasma concentration of haloperidol can be approximated by linear pharmacokinetics (2), 72 hours is an insufficient period of time for the drug to reach steady-state concentrations. For these reasons, the results of our study are not comparable to those of Van Putten et al.

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JAMES G. BARBEE, M.D.
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Effects of Pill-Giving on Maintenance of Placebo Response

SIR: Judith G. Rabkin, Ph.D., M.P.H., and associates (1) noted that pill-giving appeared unimportant for the maintenance of placebo response in a population of 50 atypically depressed patients who improved markedly after 10 days of single-blind placebo administration. Rates of relapse within 6 weeks were equal between those who continued to take placebo pills after the 10-day period and those who did not. A prospective design such as theirs is rare among studies of placebo response and therefore is a significant contribution to this body of literature.

However, it appears that they failed to control for one important variable. Those patients randomized to no further pill-taking "were told they had been taking placebo medication and that in view of their improvement it was not necessary to take active medication at that time. This information was framed positively as a fortunate outcome." The authors did not note what, if anything, patients in the other group were told.

That clinician behavior can have an important impact on placebo response is suggested by at least two studies. Park and Covi (2) found that a positive message from the physician can produce a strikingly frequent and durable placebo response even when the inert content of the pill is disclosed at the outset. In contrast to other researchers (3), Mavissakalian et al. (4) found a virtual lack of placebo response in obsessive-compulsive disorder, although it appears that they told patients the study was for the purpose of approving a drug already known to be effective (clomipramine) but not yet available in this country. Such a message may induce responder or rater bias

considering the difficulty of maintaining double-blindness under all conditions (5).

How placebo medications are presented and discussed, in practice or research, probably has a major influence on how and when patients respond to them. The question of pill-taking as one part of the placebo phenomenon may be made clear if clinician behavior can be adequately controlled.

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Reprints of letters to the Editor are not available.

APA Council Reports

At the fall component meetings of the American Psychiatric Association in Washington, D.C., on Sept. 16–19, 1992, the APA councils heard reports from their components. Following are summaries of the activities of the councils and their components.

The Council on Addiction Psychiatry

Roger E. Meyer, M.D., Chairperson

The Council on Addiction Psychiatry and its subcomponents have a strong commitment to providing psychiatric leadership in the study, prevention, and treatment of addictive disorders. The council will continue to provide recommendations to APA on research training and treatment in this field.

Over the past year the major portion of the council's attention has focused on the reorganization of the Alcohol, Drug Abuse, and Mental Health Administration (ADAMHA), its institutes and offices, funding for alcohol and substance abuse treatment, educational and certification opportunities for psychiatrists working in this subspecialty area, and clearer definition of treatment methods and services. At both its May and September meetings, the council received several distinguished visitors from the former ADAMHA and its reorganized institutes and offices. As a result of the reorganization, the National Institute on Drug Abuse (NIDA) and the National Institute on Alcoholism and Alcohol Abuse (NIAAA) report to the National Institutes of Health; the Center for Substance Abuse Treatment, the Center for Substance Abuse Prevention, and the Center for Mental Health Services now report to the Substance Abuse and Mental Health Services Administration (SAMHSA). The council conveyed concerns regarding support for education, services, and research to the leadership of these entities and offered assistance in areas of mutual agreement. The council believes that frequent contact with the heads of all of these agencies is essential if APA's informational efforts are to be coordinated, inclusive, and effective.

Support for services and professional training intersect under SAMHSA. The council urged SAMHSA leaders to coordinate funding policy and programs in a way that would ensure the development of addiction psychiatry fellowships. Cooperation among SAMHSA, its centers, and NIDA and NIAAA regarding training opportunities in both clinical and research settings was also strongly encouraged by the council.

The council registered its concern to SAMHSA and APA leadership about the decreasing interest of the private sector in supporting treatment for addictions. The council cautioned that the public sector will have to absorb a growing proportion of the patient population and urged that increased resources for services be built into federal funding mechanisms. In addition, the council believes that because the service delivery systems for alcohol and drug abuse are not necessarily the same as the basic psychiatric service system, coverage provided for alcohol and drug abuse services may be shortchanged in insurance reform legislation. Consequently, the council urged APA's leadership to include specific mention and delineation of coverage for treatment

of alcohol and other drug disorders when attempting to influence federal legislation for health insurance reform. Having worked through APA and with other physicians around the country, the council was pleased that the American Medical Association (AMA) agreed to include detoxification services in a minimum benefits package for the uninsured.

Current cuts in insurance reimbursement and increased controls by managed care entities have been particularly devastating to alcohol and drug abuse treatment programs. Reimbursement schemes frequently do not provide for adequate treatment over time, which includes detoxification, intensive residential and/or partial treatment, and appropriate aftercare. There has also been a trend to establish "all-inclusive" rates for substance abuse treatment that, in fact, do not include adequate payment for a psychiatric component within such treatment. The council urged APA's Joint Reference Committee, the Committee on Managed Care, and other components to be alert to these deficiencies.

Noting that the AMA's House of Delegates had accepted several resolutions concerning revised regulations for methadone maintenance treatment programs, the council felt that APA should express psychiatry's sentiments on regulation and administration of the programs. A statement developed by the council indicates that present methadone maintenance regulations can be improved, but the concerns that led to the generation of the initial regulations should not be lost. Those concerns had to do with minimizing the likelihood that methadone, dispensed through clinics, would be diverted into the streets and ensuring that methadone maintenance treatment would encompass a biopsychosocial approach.

In other areas, the council successfully urged the APA leadership to discourage prejudicial questions about substance abuse on applications for licensure, specialty board examinations, or other forms of certification. Concerning DSM-IV, the council forwarded its opinions on several unresolved issues, such as the use of the category of substance-induced mood, anxiety, and psychotic disorders, to the Task Force on DSM-IV.

The *Committee on Training and Education in Addiction Psychiatry*, Marc Galanter, M.D., chairperson, was pleased that the American Board of Psychiatry and Neurology (ABPN) scheduled the first examination for added qualifications in addiction psychiatry for the spring of 1993. The examination will be available to all ABPN-certified psychiatrists who have completed a fellowship in addiction psychiatry in a 12-month program approved by the Accreditation Council for Graduate Medical Education, beginning no sooner than

postgraduate year 5. For the first 5 years admission to the examination may also be accomplished through a clinical practice pathway in which the applicant spends 25% of his or her time with patients who have addictive disorders. The certificate of added qualifications will be valid for 10 years from the date of issuance.

Forty-eight fellowship positions in addiction psychiatry were filled last year. During 1992 the Accreditation Council for Graduate Medical Education declared a 1-year moratorium on recognition of further subspecialties. In light of the requirements for eligibility to sit for the examination for added qualifications in addiction psychiatry, the committee will keep a close watch on this development. In the meantime, various organizations supporting fellowships in addiction psychiatry continue to work toward delineation of curriculum essentials. The committee has a commitment to education in addiction psychiatry for all psychiatrists. In one effort to that end, the committee is crafting a position paper on the need for improved psychiatric training for combined general psychiatric and addictive disorders.

The *Task Force on Psychiatric Services for Addicted Patients* went through a metamorphosis this year. Early in 1992 the task force, chaired by Steven Mirin, M.D., submitted an outline for practice guidelines on substance abuse to the APA Steering Committee on Practice Guidelines. With encouragement from the council and steering committee, members of the task force committed themselves to

the full development of guidelines. It soon became apparent that work on the guidelines would slow work on the other objectives of the task force, and it was agreed that the work group on practice guidelines and the task force should be separate entities. As a result, the task force was reconstituted in midyear under the guidance of a new chairperson, Sheila Blume, M.D. The new task force has already developed an outline for a report that will define psychiatry's role and practice in the treatment of substance-dependent patients in relationship to other practitioners, will discuss appropriate mechanisms for payment for treatment of addicted patients, and will examine issues related to the funding of public sector programs. The task force will seek input from many other APA components as it moves toward completion of the report.

The *Task Force on Nicotine Dependence*, chaired by John Hughes, M.D., is administratively housed within the Council on Research; however, because of its topic, it also reports to the Council on Addiction Psychiatry. Similarly, the *Work Group on Practice Guidelines for Addiction Psychiatry*, chaired by Steven Mirin, M.D., reports to the Steering Committee on Practice Guidelines but informs and invites input from this council. Noting the overlap of interests of many APA components, the Council on Addiction Psychiatry believes this method of reporting increases the overall sensitivity to addiction issues in the Association without requiring duplicative efforts.

The Council on Aging

Burton V. Reifler, M.D., M.P.H., Chairperson

During 1992 Jerome Yesavage, M.D., ended his 5 years as chairperson of the council and Burton Reifler, M.D., M.P.H., began his term. Gabe Maletta, M.D., has assumed the position of vice-chairperson. The council and APA have greatly benefited from Dr. Yesavage's stewardship, and because of his unstinting and able efforts the council is well poised to head into the rest of the decade.

The council continued its active involvement with issues pertaining to geriatric psychiatry and geriatric patients in 1992. This year the Board of Trustees approved the establishment of the council's third standing committee, the Committee on the Senior Psychiatrist. The Committee on the Senior Psychiatrist, which will begin meeting in 1993, will serve as a focus point for psychiatrists contemplating retirement and will ensure that the knowledge base of senior psychiatrists is preserved and made available to younger members as an important resource in years to come. The committee will also provide a means of direct input into APA for the nearly 5,000 members of APA's Lifers group.

In 1991 the Board of Trustees approved the establishment of the Committee on the Ethnic Minority Elderly, in response to council recommendations that APA pay careful attention to the specific needs of this population. The committee has an ambitious agenda and will be an important resource for the council and APA in the years ahead.

The council also received approval last year to establish the Work Group on Model Standards for Private Long-Term Care Insurance. The work group met twice in 1992 and is well on its way to completing its charge to recommend to the Board and APA methods of ending discrimination against current and former psychiatric patients in the purchase of private long-term care insurance policies.

The Task Force on Models of Practice in Geriatric Psychiatry has completed its report titled "Selected Models of Practice in Geriatric Psychiatry," and this year the Board approved the report for publication. The task force is now working with American Psychiatric Press, Inc., (APPI) on final drafts of the report, which is expected to be pub-

lished before the 1993 annual meeting. The council also expects final publication of two other reports early in 1993.

Council members continue their extensive collaboration with other APA components and offices and with other medical organizations. For example, the Council on Aging continues its participation with the Council on Psychiatry and Law in the intercouncil Subcommittee on the Impaired Driver. The council expects to establish formal liaison with the Work Group on Codes and Reimbursement in late 1992, and it continues to work closely with the Council on Medical Education and Career Development to monitor issues related to education and training in geriatric psychiatry. The Council on Aging also continues its long-standing close working relationships with APA's Division of Government Relations and with the National Institute of Mental Health (NIMH).

Council members routinely provide expertise and advice on complex federal laws and regulations governing the care of the elderly in such areas as Medicare and Medicaid policy affecting older patients, nursing home law and regulations, psychopharmacologic issues affecting older patients, and Medicare payment issues.

In response to a coordinated effort by the Council on Aging and the APA Division of Government Relations, the U.S. Congress approved and the President has signed into law legislation to facilitate federal assistance to programs for education and training in geriatric psychiatry under the Public Health Services Act.

Members of the Council on Aging were active in conveying to their federal elected officials and federal regulators their concerns about the impact on geriatric psychiatry of the resource-based relative value scale (RBRVS) Medicare fee schedule; this process is ongoing. The members of the Committee on Long-Term Care and Treatment for the Elderly have been particularly effective in assisting the APA Division of Government Relations in commenting on, and obtaining changes to, proposed federal regulations governing the general care and treatment of nursing home residents and, in particular, the use of psychotropic medications with the nursing home population.

Virtually all council components met during the 1992 annual meeting in Washington, D.C., as they did again during the 1992 fall component meetings. The council will convene for its January 1993 meeting in Washington, D.C., where members will again meet with key Congressional staff and with members of Congress. The January meeting provides a unique opportunity for the members of the council to develop personal and professional relationships with Congressional staff and members of Congress directly involved in the development of federal policy affecting geriatric psychiatry and other federal issues of concern to the council.

At the January 1993 meeting, council members and chairpersons of components will also undertake a thorough review of the structure of the Council on Aging and all components, and they will consider what, if any, changes are required in that structure to ensure that the council continues to respond to the specific needs of geriatric psychiatry and our patients.

The Council on Aging and its components look forward to continuing their work in 1993 and to enhancing APA's role in the field of geriatric psychiatry.

The *Committee on Long-Term Care and Treatment for the Elderly*, Ira Katz, M.D., Ph.D., chairperson, and the *Task Force on Reimbursement Options and Alternatives in the Care of Older Persons*, Howard Goldman, M.D., chairperson, continue their practice of holding joint meetings. Members of the committee and the task force provide expert assistance to APA's Division of Government Relations on issues related to nursing home care and to federal reimbursement for psychiatric services under the Medicare and Medicaid programs, and they have participated in consensus conferences on the use of medications in nursing homes.

In response to the recommendations and efforts of committee member Barry Fogel, M.D., Senator John Chafee (R-R.I.) and Representative Ronald Machtley (R-R.I.) introduced legislation to repeal the discriminatory 50% copayment requirement for psychiatric services provided to Medicare nursing facility patients.

The *Committee on the Ethnic Minority Elderly*, Kenneth Sakaue, M.D., chairperson, was approved as a permanent committee by the Board of Trustees in 1991, following completion of the report of the Task Force on the Ethnic Minority Elderly. The committee met during the APA annual meeting and also during the 1992 fall component meetings.

The committee is now addressing issues related to racism and racial polarization as they affect minority communities, and it proposed holding a workshop at the annual meeting in 1993. The committee will also collect and disseminate information on model programs for ethnic elders.

The *Task Force on Models of Practice in Geriatric Psychiatry*, Marion Goldstein, M.D., chairperson, completed work in 1992 on its report titled "Selected Models of Practice in Geriatric Psychiatry." The report, which was approved for publication by the Board, is now undergoing the APPI editorial process, and publication is anticipated in early 1993.

Given the increasing importance of geriatric psychiatry, the Council on Aging has recommended the establishment of a permanent Committee on Practices in Geriatric Psychiatry. As of this date, the proposed new committee has not yet been approved, but the council hopes that APA will grant its request for the new committee in 1993.

The *Work Group on Model Standards for Private Long-Term Care Insurance*, Barry Fogel, M.D., chairperson, was approved by the Board of Trustees in 1991 and was established to develop and recommend policy standards aimed at eliminating long-term care insurance discrimination against individuals requiring treatment for mental illness. Current insurance standards governing the issue are extremely weak. The work group held two meetings in 1992 and is well on its way toward meeting its charge.

The *Task Force on the White House Conference on Aging*, Sanford Finkel, M.D., chairperson, has been hampered in its work by the on-again, off-again target date for the next decennial White House Conference on Aging. It appeared that a conference would definitely be set for 1993, but late in 1992 prospects became remote. Congress, however, has authorized a White House Conference on Aging in 1994, and it is hoped that there will be renewed efforts in the executive branch to meet that target.

In the interim, Dr. Finkel, together with council member Donald Hay, M.D., joined with representatives of the psychology, social work, and nursing professions to develop an agenda for a mental health mini-conference to be held before the next White House Conference on Aging. With support from NIMH, representatives of the four disciplines met twice in Washington to review the record of the last White House Conference on Aging (in 1981) and to prepare a paper for NIMH on the progress toward meeting the mental health goals adopted by that conference. This paper, which is preparation for the mini-conference, is now undergoing final revisions and will provide important guidance for representatives of the mental health care community in developing an agenda for the next White House Conference on Aging.

The *Task Force on Education and Training in Geriatric Psychiatry*, Charles Shamoian, M.D., chairperson, finally saw the fruition of its major federal legislative initiative: the expansion of federal support for programs of education and training for geriatric psychiatry. To that end, Congress approved and the President signed into law legislation to amend Title VII of the Public Health Service Act to expand opportunities for fellowship funding for training in geriatric psychiatry and to permit departments of psychiatry to be loci of geriatric training.

The *Jack Weinberg Memorial Award Board*, Gabe Maletta, M.D., chairperson, is a new board established within the council in response to the request of the Council on Internal Organization. The Council on Aging agreed to resume full responsibility for the administration of the Jack Weinberg Memorial Award in Geriatric Psychiatry commencing with the award to be given at the 1993 annual meeting. The Weinberg award for 1992 was awarded to Dan Blazer, M.D.

The Council on Children, Adolescents, and Their Families

Mohammad Shafii, M.D., Chairperson

The work of this council and its components continues to be directed toward maximizing the effectiveness of APA in addressing the mental health needs of children, adolescents, and their families. This charge has been carried out through workshops, reports, position statements, and liaison with other APA components and allied organizations. The council has kept abreast of many issues that affect its

patient populations through intramural and extramural liaison activities.

The council continues to keep an eye on the continuation of increased funding for children and adolescents. In 1990-1991 there was a 20% increase in funding for child psychiatric research. This year, with encouragement from the council, APA President Dr. Lawrence Hart-

mann corresponded with Dr. Frederick Goodwin, Director of the National Institute of Mental Health (NIMH), and learned that NIMH is planning a 14%–16% increase in research on children and adolescents.

In addition to the amount of money allocated to child and adolescent mental health facilities, the council plans to look into the kinds of services that are being provided. Currently prevalent are community-based programs, an offshoot of the deinstitutionalization movement of a decade ago. These programs allow for the treatment of larger numbers of the less severely mentally ill. The question is what kind of services and resources will ultimately be supported. The numbers of children and adolescents requiring hospitalization services have been upwardly revised.

The council found the proposed Oregon plan regarding health care to be concrete and specific. This plan is committed to mandated mental health coverage and increased access to health care while based on the premise that there are limited resources.

The Council on Accreditation of Services for Families and Children requested that APA send a representative to that council's board of trustees. Dr. Shafii had asked Dr. Work to look into the matter. Dr. Work recommended that APA send a representative to the council's board of trustees because it is important that APA work with the council to develop standards for accreditation of family and child services nationwide.

Mary Jane England, M.D., and Leonard Sacks, Ph.D., described the work of the Mental Health Services Program for Youth, which is funded by the Robert Wood Johnson Foundation. This program includes a systems approach to corrective solutions for coordinating the various services for children and youth. By working with state social welfare, juvenile justice, and educational systems, this program can assess conflicts and differences and can help improve the care provided. Because of the way the programs are organized within the states, even the children who are most seriously distressed do not receive coordinated, effective care. Council members voiced their concern that psychiatrists are not adequately involved with the projects. The council is considering approaching NIMH to offer to perform research on services.

Dr. Carolyn Robinowitz summarized the search committee's progress in its quest for a Director for the Office of Minority/National Affairs. She thanked Ms. Gail Nelson for her work with the council and commented on the consultation provided by Dr. Henry Work. Dr. Sabshin explained that he will introduce to the Board of Trustees the idea that a minority individual be hired as Director of the Office of Minority/National Affairs, and in light of budget constraints this individual might also serve as Director of the Office of Psychiatric Services. The added responsibilities might require the search committee to take into account the need to tailor the requirements for this position to include experience and expertise in public psychiatry.

Dr. Marshall Forstein, chairperson of the Commission on AIDS, joined the council for a discussion of the AIDS epidemic as it concerns children and adolescents. More studies and research are needed, as are referral services for families currently dealing with AIDS. Dr. Forstein advised that during the HIV infection period referrals be made to family psychiatrists and to child psychiatrists in cases where children are forced to cope with neurodevelopmental difficulties caused by HIV infection.

The council discussed development of subspecialty boards, particularly the American Board of Adolescent Psychiatry. Council members felt that younger people going through training would be more likely to go into child and adolescent psychiatry, not adolescent psychiatry alone, because certification in child and adolescent psychiatry provides a wider scope of practice. After further discussion, the council decided to drop the issue.

The proposed neurodevelopmental pediatrics subspecialty, its curriculum, and its impact on patient care were discussed. The council has kept an open mind regarding communication with other specialties and has not allowed territorial issues to cloud the discussions with these specialties.

When the council met with the Joint Commission on Government Relations, Dr. Shafii asked about establishing a liaison from the council to the commission. Although the commission is sympathetic to the council's wishes, it does not have the personnel to spare for such a liaison. The council then suggested that since there are child psychiatrists on the joint commission, perhaps one of them could act as liaison

to the council. Staff suggested that the council stay in touch with district branches because much of the child health care legislation is handled at the state level. The council requested that its members be added to the mailing list for legislative newsletters produced by the Division of Government Relations.

Dr. David Pruitt spoke about practice guidelines and health reform packages being developed by APA. Both APA and the American Academy of Child and Adolescent Psychiatry are developing guidelines on eating disorders and depression. There was a discussion of whether or not children and adolescents should be included in the APA guidelines or whether that should be left to the American Academy of Child and Adolescent Psychiatry. After extensive discussion, the council agreed that the APA Steering Committee on Practice Guidelines should include children and adolescents when developing various guidelines, including those on depression. The precedent established by the inclusion of criteria for children and adolescents in DSM-III, DSM-III-R, and DSM-IV should be followed.

As a result of an administrative reorganization of APA awards, the council now oversees the Blanche F. Ittleson Award for Research in Child Psychiatry and the Agnes Purcell McGavin Award. At issue was the council's role regarding these awards. After considerable discussion, the council concluded that although the choice of award recipients is the responsibility of the award boards, the council, in addition to other sources, should be asked by these boards to submit formal recommendations and should have the right of accepting or rejecting the award boards' selections.

The council nominated five child and adolescent psychiatrists to fill two vacancies on the child and adolescent psychiatry committee of the American Board of Psychiatry and Neurology. Their names were submitted for approval to the Joint Reference Committee and, subsequently, to the Board of Trustees.

The *Committee on Chronically Ill and Emotionally Handicapped Children*, Kendon W. Smith, M.D., chairperson, proposed that Area councils and district branches be asked to sponsor meetings and workshops on the needs of the chronically mentally ill child. A second request is for Area councils and district branches to identify the fiscal consequences of the new child Supplemental Security Income regulations state by state and to communicate their findings to psychiatric practitioners through a mailing to members or through the district branch newsletters. Dr. Smith also asked the council to suggest to NIMH that proposals be requested for epidemiologic study of children and adolescents who are psychotic, in foster care, or in correctional settings.

The *Committee on Family Violence and Sexual Abuse*, Sandra Kaplan, M.D., chairperson, submitted the final version of the book *Family Violence: A Clinical Guide* for the council's approval. The council submitted the manuscript to the Joint Reference Committee, which deferred action until its February 1993 meeting. The comments of the primary reviewers were sent to Dr. Kaplan, who will make the necessary revisions in time for the Joint Reference Committee to receive the final draft by the first week in January.

The *Committee on Juvenile Justice Issues*, Richard C. Marohn, M.D., chairperson, will prepare a monograph that will review the history of the relationship of psychiatry and juvenile delinquency. The committee hopes to have the manuscript ready for APA's 150th anniversary in 1994.

Ms. Julie Schroyer, Assistant Director of the Division of Government Relations, asked for the committee's support for reauthorization of the Juvenile Delinquency Prevention Act, which she hopes will incorporate mental health services for incarcerated juveniles.

Dr. Michael Kalogerakis represented APA at the annual meeting of the National Coalition for the Mentally Ill in the Criminal Justice System. Dr. William Buzogany and other child psychiatrists will represent the interests of child psychiatry on the coalition's juvenile justice and juvenile delinquency prevention panel.

At the 1993 APA annual meeting Dr. Marohn will try to organize a caucus of APA members who are involved in juvenile justice issues in order to reach a wider group of like-minded psychiatrists and to obtain grass-roots support for and participation in the work of the committee.

The *Committee on Psychiatry and Mental Health in the Schools*, Irving H. Berkovitz, M.D., chairperson, requested the approval of the committee's monograph, *Psychiatric Consultation in the Schools*, for

publication. The council and the Joint Reference Committee did approve the monograph, with minor changes to be incorporated before submission to the Board of Trustees. Also, Dr. Berkovitz requested permission for the committee to develop a brochure based on this monograph. The council asked the committee to submit information on the content of the brochure, the costs involved, the target audience, and a proposal for seeking funds.

The committee had tried unsuccessfully to stimulate the interest of the district branches in mental health consultation by sending articles to each district branch newsletter, but only one response was received. Dr. Berkovitz asked whether the council thought it would be beneficial to work through the American Academy of Child and Adolescent Psychiatry since 80% of the psychiatric school consultants are members of both APA and the American Academy of Child and Adolescent Psychiatry.

The *Task Force on Day Care for Pre-School Children*, Myron Belfer, M.D., chairperson, submitted a manuscript titled "Day Care for Early Pre-School Children: Implications for the Child and Family," which the council and the Joint Reference Committee approved with minor revisions. This will be submitted to the Board for final approval.

Since the task force had accomplished all but two of its tasks, the council recommended to the Joint Reference Committee that this task force be terminated, and it requested that a standing committee on day care of preschool children be established in its place. After considerable discussion, the Joint Reference Committee approved the council's recommendation, with the proviso that the role of this proposed committee be expanded to cover not only day care but preschool children as a whole. The charge to the Committee on Pre-School Children would be to do the following.

1. Review data on the prevalence of and risk factors for mental health problems in infants, toddlers, and preschool children.

2. Determine and implement mechanisms to support the efforts of psychiatrists and other professionals who seek to prevent and treat such problems.

3. Monitor developments in governmental policy that affect the mental health needs of young children and work for policies that are in the best interest of these children.

4. Explore possible mechanisms for promoting good parenting practices, including the creation and development of high school and college curricula on parenting that would emphasize normal growth and development and the role of the family, relatives, neighbors, the workplace, and the community.

5. Establish contacts with other organizations to garner support for the development of a national policy on affordable, high-quality day care. Such efforts would include the following actions.

- a. Seek support, through the district branches, for the development of high-quality day care at the local and state levels.

- b. Develop criteria with which parents could seek high-quality day care for infants, toddlers, and children.

- c. Develop criteria for determining when children in day care need mental health evaluation.

- d. Determine factors in preschoolers related to risk for later development of substance abuse and psychiatric disorders.

- e. Develop information about special procedures that can (or should) be used by day care personnel to best care for children with emotional needs related to particular identifiable situations (e.g., abandonment, abuse, trauma, families with psychiatric disorders).

- f. Continue to review research on day care and report to the Council on Children, Adolescents, and Their Families, the Joint Reference Committee, and the Board of Trustees.

6. Establish contacts with APA components and other organizations to engender support for the development of policy and funding for the diagnosis, treatment, and rehabilitation of infants, toddlers, and preschoolers who are victims of parental abuse of drugs and alcohol or suffer from AIDS.

The *Task Force to Study the Use and Abuse of Hospitalization of Adolescents*, Elizabeth B. Weller, M.D., chairperson, received support from the Kenworthy-Swift Foundation and was able to meet for the first time in September. Mr. Christopher Dykton, the part-time research assistant for the task force, has been working with Dr. Weller and Dr. Robert Hendren to gather data from state regulatory and advocacy groups.

In conjunction with the annual meeting of the American Academy of Child and Adolescent Psychiatry, the task force will have a presentation titled "Hospitalization of Children and Adolescents: Dilemmas and Guidelines," which will deal with the different aspects of misuse and abuse of hospitalization. The task force will also write a monograph on this topic.

The *Task Force on the Psychiatric Aspects of New Reproductive Technologies*, Miriam Rosenthal, M.D., chairperson, is working on the final draft of its report. The task force did not meet in September because of current budget constraints, but the members will continue to work by means of conference calls and correspondence.

The Council on Economic Affairs

Robert Gibson, M.D., Chairperson

The activities of this council and its components during 1992 reflect APA's continued efforts to influence the many commercial and governmental entities that affect the provision of psychiatric care. Members of the council and its components maintained liaison with representatives of such organizations as the Social Security Administration, the Joint Commission on Accreditation of Healthcare Organizations (JCAHCO), the Health Care Financing Administration (HCFA), and major managed care and insurance companies.

The *Committee on Financing and Marketing*, Robert O. Friedel, M.D., chairperson, has focused on marketing assistance for APA members, primarily through the district branches. Its major concerns include negative trends in corporate response, decreasing insurance benefits for psychiatric care, inappropriate utilization management activities, and a blurring of the roles of psychiatrists and psychologists. The committee has set the following goals for the year: 1) up-

dating the APA marketing manual for district branches, 2) reviewing and editing a new chapter on marketing psychotherapy for inclusion in the manual, and 3) presenting a workshop on total quality management for the 1993 annual meeting.

The *Committee on Hospital-Based or Hospital-Related Services*, Boris Astrachan, M.D., chairperson, focused its attention on the initiatives of the JCAHCO, including its "Agenda for Change," major revisions to all standards manuals, and the formation of expert task forces related to the development of mental health indicators. It participated in the JCAHCO field review process by providing comments and suggestions relating to proposed new or revised standards contained in the *Accreditation Manual for Hospitals* and the *Consolidated Standards Manual*.

The committee also brought to the attention of the council and the APA leadership its concerns regarding the "demedicalization" of the

JCAHCO, the decreased reliance on professional and technical advisory committees and physicians, and the increased reliance on expert panels. Additional concerns regarding flaws in the field review process were articulated.

The committee was supportive of the efforts of APA President Dr. Joseph English to discuss APA concerns with leaders of the American Medical Association (AMA) and provided suggested agenda items for a meeting with AMA leaders and those who serve on behalf of the AMA on the JCAHCO Board of Commissioners.

The *Committee on Interprofessional Affairs*, William Webb, Jr., M.D., chairperson, continued its work with the Joint Commission on Interprofessional Affairs, which comprises representatives of the American Nurses' Association, the American Psychological Association, the National Association of Social Workers, and APA. In the spring of 1992 a joint commission leadership conference was held. The elected leaders, chief executive officers, and selected staff participated. Resulting from that conference and subsequent meetings of the chief executive officers was a substantial change in the ways in which the four organizations will continue their liaison.

Sponsored by the American Psychological Association, a resolution was offered to change the structure of the Joint Commission on Interprofessional Affairs by eliminating the role of commissioners. The resolution stated that the two commissioners appointed by each organization often lack the authority to speak or act on behalf of their associations. Although important initiatives have been identified and pursued by the commissioners, progress on them has been slowed by the concurrence processes. Additionally, the cost of this form of liaison was judged to be excessive.

The resolution was brought to the governing bodies of each organization and received approval. Consequently, the liaison activities of the four organizations are now carried out through quarterly meetings of the chief executive officers and an annual meeting of elected leaders, executives, and appropriate staff.

The *Committee on Managed Care*, Steven Sharfstein, M.D., chairperson, continues to address issues for psychiatry and managed care through support of legislation and regulation, research, education, and dialogue with the managed care industry. Special projects this year included publication of the proceedings of the Conference on Ethics in Managed Care for Psychiatry, an annual meeting workshop on psychotherapy in managed care, continued work with the network of district branch managed care liaisons and initiation of local efforts in managed care, and continuation of a series of meetings with managed care organizations to discuss problems documented through APA's 800-number telephone service and to seek redress at the policy level. New projects include review of criteria for medical necessity, network development, and the assessment of patient referral and triage methods used by the managed care companies. The committee continues to support managed care regulation activities and the review and presentation of cases documented through the 800-number (1-800-343-4671) telephone service.

The *Committee on Occupational Psychiatry*, Peter L. Brill, M.D., chairperson, has focused on issues of importance to the APA membership, including workplace stress, the Americans With Disabilities Act, and organizational consulting. The committee will present a component workshop on the opportunities for psychiatry under the Americans With Disabilities Act. The committee further reported that the Academy of Occupational and Organizational Psychiatry will meet in January 1993 and at the APA annual meeting in May.

The *Committee on Private Practice*, Peter Kramer, M.D., chairperson, continues to express its strong concerns about the problems currently facing the psychiatrist in private practice. As most of the current identified problems have potentially severe economic consequences for the private practitioner, the committee sought and received a transfer from the Council on Psychiatric Services to the Council on Economic Affairs.

At its first meeting with the council, the committee articulated its belief that APA leaders may not be fully aware of and sensitive to the serious problems that confront the psychiatrist in free-standing private practice. The council responded by detailing the committee's concerns at meetings of the Joint Reference Committee and the Board of Trustees.

The council will continue to work closely with the Committee on Private Practice and offer the assistance it can to address the challenges faced by this group of practitioners.

The *Committee on Quality Assurance*, Marlin Mattson, M.D., chairperson, reviewed and critiqued various criteria, standards, and screens affecting psychiatric treatment that are published by other organizations and the federal government. A great deal of effort has focused on review of the HCFA generic quality screens for hospital inpatient care. Recommendations were developed and forwarded to the HCFA for its consideration, and the committee will continue to work with the HCFA to bring about meaningful revisions in the screens.

The committee's work with the HCFA has also included exploration of the possibilities of participating in the development of review criteria based on practice guidelines on depression. The committee believes this may be one of the most important opportunities APA has been offered for participating in a quality improvement initiative that may play an important role in the evolving health care arena. As the details of potential collaboration become more concrete, the committee will seek approval of the council and the Board for APA involvement.

The *Committee on Universal Access to Health Care*, Herbert S. Sacks, M.D., chairperson, refined its mission and planned its agenda. The committee's tasks are 1) to disseminate information to the membership relating to universal access to health care, through articles in APA publications and through programs at the annual meeting and regional meetings, 2) to develop a knowledge base and keep abreast of developments in health care economics as they pertain to universal access to care, 3) to study global issues involving health care delivery systems, 4) to serve as a resource to the Board of Trustees as it develops APA policy on universal access, and 5) to interact with other APA components, both to receive relevant input and to provide consultation as indicated. The committee's specific activities have included preparation of a bibliography identifying approximately six publications on universal access and submission of a number of articles to *Psychiatric News* on topics such as developments in the states, international developments, and Congressional actions.

The *Task Force on Prospective Payment Issues*, Joseph T. English, M.D., chairperson, continued to monitor payment issues and to explore potential alternative payment mechanisms. Among these efforts was a review of National Institute of Mental Health studies of federal legislative and administrative activities related to prospective payment for psychiatry. The task force believes there are no immediate threats to psychiatry's exempt status, and it continues to work closely with the Prospective Payment Assessment Commission as it works to improve reimbursement under the Tax Equity and Fiscal Responsibility Act of 1982 (TEFRA). The task force is monitoring several innovative payment reform initiatives underway in the states. These are being watched closely by the federal government and the field.

The *Work Group on Codes and Reimbursement*, Chester Schmidt, Jr., M.D., chairperson, has had a very busy year. Major issues that the work group has dealt with include the new evaluation and management codes published in the 1992 *Physicians' Current Procedural Terminology* (CPT), the Medicare fee schedule, and the lack of adequate CPT codes for psychiatry. Members of the component have presented workshops on the new codes and the new Medicare fee schedule to several district branches. In addition, seminars and workshops were presented at the Joint State Legislative and Public Affairs Institute, the APA Assembly, and the annual meeting. The work group will again sponsor a workshop on the evaluation and management codes and a course on the basics of CPT coding at the annual meeting in San Francisco. The work group has also recommended an additional psychotherapy code and some revisions to existing codes for the Assembly's approval and submission to the AMA CPT editorial panel. Additionally, Dr. Schmidt has submitted the document *CPT Handbook for Psychiatrists: Coding for Maximum Reimbursement* to American Psychiatric Press for publication.

The *Work Group on the Harvard Resource-Based Relative Value Scale (RBRVS) Study*, Donald Scherl, M.D., chairperson, continues to address remaining problems contained within the new RBRVS Medicare fee schedule. Although the RBRVS was expected to result in an increase in payments for "cognitive" services, including psychiatry, the new Medicare rates for psychiatric services actually are damaging to urban-based psychiatry.

Many of the numerous statistical, methodological, and conceptual flaws contained in the proposed RBRVS for psychiatry were ad-

dressed, and some corrections have been made through APA's intensive lobbying effort and the work group's high-level negotiations with the HCFA.

APA and the work group testified before the several relevant committees of the U.S. House of Representatives and Senate, submitted initial and final comments on the proposed rule, then submitted comments on the final rule, communicated APA concerns to all 535 mem-

bers of Congress, and coordinated a massive APA grass-roots letter-writing campaign. Additionally, APA and the work group have met throughout the year with senior HCFA staff, staff of the U.S. Department of Health and Human Services, and Congressional office staff to express psychiatry's concerns with the new fee schedule and to discuss how the fee schedule might be changed to address those concerns.

The Council on Internal Organization

Thelissa A. Harris, M.D., Chairperson

The council oversees "all those matters involved in the organizational structure of the Association not covered by constitutional committees." Eleven components report directly to the council, and six other components report to committees under the council: four award boards report to the Committee on Grants and Awards, and a subcommittee and a task force report to the Scientific Program Committee. Components of the council cover many areas of responsibility: 1) annual meetings—including program content, exhibits, and arrangements; 2) information systems—including data processing and telecommunications; 3) benefits—including benefits offered to members as well those for APA staff; 4) maintenance of the APA headquarters building; 5) advertising policy; and 6) the federation of auxiliary organizations. Following are the highlights of council and component activities over the past year.

The *Committee on Information Systems*, Zebulon C. Taintor, M.D., chairperson, learned that APA now has 60 AST personal computers. All of the departments that were slated to receive personal computers now have them: Office of Membership, Office of Economic Affairs, Division of Government Relations, Office to Coordinate the Annual Meeting, H&CP Service, Medical Director's Office (which includes the Office of Psychiatric Services), and the Library/Archives. Three more departments are scheduled to receive personal computers in January 1993: Division of Public Affairs, Office of Education, and Office of Meetings and Exhibits Management.

The Committee on Information Systems has proposed two days of presentations, including two 3-hour sessions, for the 1993 annual meeting.

Following is a summary of the 1992 activities of the *Committee on Telemedical Services*, Frank W. Brown, M.D., chairperson.

1. *Health Care Financing Administration (HCFA)*. A demonstration program to assess the feasibility of reimbursement for consultative services provided to Medicare beneficiaries has been suggested by the U.S. House of Representatives. The committee supported such a project, suggesting possible sites and offering to provide any appropriate assistance. Members also met with the HCFA to explore important underserved and technically feasible areas for telecommunications, i.e., prisons and certain interrelated institutions.

2. *Article in Hospital and Community Psychiatry journal*. The article "Using Telemedicine to Improve Health Care in Distant Areas" by Jane Preston, M.D., Frank Brown, M.D., and Betty Matley, M.L.S., was published in the journal in January 1992. The article reviewed the historical development of telemedicine, practical applications, medical-legal concerns, and ethical considerations.

3. 1992 annual meeting programs

a. A committee exhibit, under the direction of Dr. Preston, Dr. Jonathan Lieff, and Dr. Brown, used audio/video telecommunication equipment and interactive patient interviews. The participants observed the use of telecommunication in the delivery of health care services and were given an opportunity to use the equipment.

b. The Workshop on Telemedicine in Psychiatric Practice, chaired by Dr. Brown, provided an opportunity for participants to understand applications of interactive medicine, as well as ethical and medical-legal considerations.

4. *Cost feasibility of using telecommunications*. A pilot project to study using telecommunications instead of in-person meetings for administrative and educational APA activities is being considered. It does not seem cost-effective for the Association to use telecommunications to replace in-person meetings at this time.

The *Scientific Program Committee*, John M. Oldham, M.D., chairperson, is largely responsible for the fact that the last four annual meetings have been marked by a high level of member satisfaction and success by almost every measure. The last three meetings were the largest in APA history. This is a reflection of the importance of continuing education to our members and the fact that the APA annual meeting remains the foremost meeting in the world for the presentation of the most up-to-date information in the field. Following are some committee highlights for 1992.

1. *Industry symposia*. New procedures for evaluating industry symposia were implemented this year, with the purpose of increasing APA's oversight. An annual meeting with the industry representatives who plan the symposia has improved communication between APA and industry.

2. *Scientific exhibits*. The Subcommittee on Scientific and Educational Exhibits was disbanded, since the scientific program has expanded to include many of the areas previously included in these exhibits—new research, oral/slide presentations, and poster sessions. However, the meeting continues to have educational exhibits submitted by nonprofit organizations. These are reviewed by the Committee on Advertisers and Exhibitors.

3. *Subcommittee on Film and Subcommittee on Video*. These two subcommittees were combined. Videotape technology has evolved so much that the availability of 16-mm films has diminished. Therefore, the film program at the annual meeting became more difficult to organize each year, while the video program grew. Merging the two subcommittees allows for one subcommittee meeting per year instead of two, reducing the costs of travel, film rentals, audio/visual equipment, and labor for the annual meeting. The chairpersons of the combined subcommittees will serve as cochairpersons of the new subcommittee until their terms expire in 1993.

4. *Hearing-impaired individuals*. Because of the passage of the new Americans With Disabilities Act, APA will need to ensure that funds are available to provide assistance to disabled persons wishing to participate in our annual meetings.

The *Committee on Advertisers and Exhibitors*, Maurice J. Martin, M.D., chairperson, proposed a study of the flow of all events during the annual meeting. This was prompted by 1) concerns of the allied mental health organizations that traditionally meet in conjunction with the meeting, 2) the shift of the Opening Session and Business Meeting to Sunday from Monday, and 3) a request from exhibitors to change

the hours for the exhibits hall. An analysis of the current flow of the meeting shows that meeting participants are arriving earlier.

The study will also consider travel industry trends encouraging a Saturday night stay-over and the impact any changes might have on the Assembly of District Branches, the Business Meeting, and meetings of other governance bodies.

The council asked staff to contact relevant groups and obtain information concerning the schedule of the annual meeting, requesting that responses be received by the end of the year. The respondents should be asked to consider educational advantages along with financial advantages. The council will act as arbitrator, possibly having a conference call after the data have been collected.

Lucy D. Ozarin, M.D., chairperson of the *Headquarters Committee*, restated the committee's charge as the responsibility to 1) survey and ensure general care of the building, 2) oversee use of space, and 3) review requests from outside organizations.

The council expressed its concern that the headquarters building has no sprinkler system. The building codes did not require such a system at the time the building was built. Staff looked into installing such a system 3-4 years ago, including asking for bids, but installation was prohibitively expensive. The total infrastructure cost was estimated to be \$528,044.

The committee also stated that JBG, the building manager, has contracted to survey the building for compliance with the Americans With Disabilities Act.

The *Committee on Special Benefit Programs*, Eva V. Ebin, M.D., chairperson, reported that all special benefit programs were doing very well. The retirement program has been successful, particularly with Members-in-Training. The Vantage Travel program has had limited enrollment and is considering trips of shorter length to accommodate more APA members' vacation plans.

The following programs are being looked into as possible benefits for members:

1. A second mortgage loan program.
2. A Gold MasterCard for residents.
3. A new series of tax seminars.
4. Discount office products and equipment.
5. Discount telephone service.
6. Discount automobile insurance.

Professional Risk Management Services, Inc., (PRMS) currently administers and staffs the APA special benefit programs in addition to serving as administrator for the APA-sponsored insurance programs. The Council on Internal Organization approved the committee's recommendation that in May 1993 the special benefits programs and the committee be transferred from PRMS, thus becoming an APA in-house function.

The legal seminars and the legal consultation plan are risk management and insurance-related functions, financially supported almost entirely by the Psychiatrists' Purchasing Group, Inc. These two programs were transferred to the purchasing group for oversight and administration.

Martha J. Kirkpatrick, M.D., chairperson of the *Committee on History and Library*, reported on the funding drive for the rare book room to be housed in the APA library. Approximately \$12,000 has been raised.

Plans for the Sesquicentennial include a special Benjamin Rush lecture, a possible compilation of presidential addresses from 1969 to 1994, and a display of images of madness.

Following is a summary of the activities of the *Committee on Grants and Awards*, Mary Jane England, M.D., chairperson.

1. Reorganization of awards

- a. The Board of Trustees asked the committee to review the awards program, specifically, the possibility of placing awards under the oversight of appropriate councils, e.g., placing awards dealing with children under the Council on Children, Adolescents, and Their Families. The following awards have been transferred from the Council on Internal Organization:
 - Blanche F. Ittleson Award and Agnes Purcell McGavin Award, to the Council on Children, Adolescents, and Their Families
 - Marie H. Eldredge Award, to the Council on Psychiatric Services

- Jack Weinberg Memorial Award for Geriatric Psychiatry, to the Council on Aging
- Isaac Ray Award in Memory of Margaret Sutermeister and the Manfred S. Guttmacher Award, to the Council on Psychiatry and the Law

- b. The policy of the Committee on Grants and Awards has been to decentralize the awards program. The individual award boards or committees have selected their winners independently, under the general oversight of an appropriate council. It is the hope of the committee that any concerns expressed by the council or other committees relative to a proposed award recipient can be handled informally with the award component. However, since there is a timetable for selection of award winners (the September meetings of the components for awards that include lectures), a final arbitrator is necessary. The committee recommended that this final arbitrator should be the Board of Trustees. This action was passed by the Board in September 1992.

- c. In addition, the committee feels that development of a strategic approach for the awards program, viewing each year's award winners en masse, might help serve other functions of the Association, such as public affairs and government relations.

2. *Reorganization of the Convocation program.* To streamline the Convocation program, the committee and the council agreed that each award should be presented at the time of the lecture instead of during the Convocation. This means that the Administrative Psychiatry Award, Oskar Pfister Award, and Seymour D. Vestermark Award will no longer be presented during the Convocation. Recipients of these awards will be listed in the Convocation program and invited to any reception that is held. Alternative occasions for presentation of other awards that are now presented during the Convocation are being considered.

3. Changes in specific awards

- a. *Samuel G. Hibbs Award.* This award, which included an annual meeting lecture, is no longer funded by Anclote Manor Hospital and thus was dropped by the Association. The award, established in 1981 to honor Samuel G. Hibbs, M.D., former president of Anclote Manor Hospital, was given to a person or team for an unpublished paper on a clinical subject.
- b. *Oskar Pfister Award.* Dr. George T. Harding IV contributed \$10,000 to help support this award for work in psychiatry and religion. The contribution (on behalf of Dr. Harding, his nephew, Dr. Herndon P. Harding, Jr., and his brothers, Drs. Richard and Herndon Harding) is a memorial to his grandfather (George T. Harding II, M.D.), his father (George T. Harding III, M.D.), and his uncle (Charles W. Harding, M.D.).

- c. *American Psychiatric Association/Psychiatric Institutes of America (APA/PIA) Foundation Award for Research Development in Hospital Psychiatry.* The award board believes that the small number of applications for this award in past years is due to its narrow focus. Therefore, the criteria for the award were broadened to include mental health services and systems research. The council also approved changing the award name from the APA/PIA Foundation Award for Research Development in Hospital Psychiatry to the APA Award and Fellowship for Research Development in Psychiatric Services Research.

- d. *Benjamin Rush Lecture.* Upon the recommendation of the Committee on History and Library, the council approved an increase in the honorarium for this lecture, from \$500 to \$1,000. The nonmember expenses will continue to be provided for, at \$500.

4. *Funding for new awards.* The "Policies and Procedures for Awards," approved by the Board of Trustees in December 1986, stated that a lump sum donation of at least \$25,000 is required to fund a new award. The council approved raising this amount from \$25,000 to \$50,000.

5. *Proposal for George Tarjan Award.* On the recommendation of the Committee on International Medical Graduates, the council approved the new George Tarjan Award, designed to recognize contributions to the integration of international medical graduates into

American psychiatry. An amount of \$50,000 is needed to fund the award.

The *Committee on Human Resources*, Jack W. Bonner III, M.D., chairperson, discussed the following.

1. *Staff health insurance plans*. APA has the MD-IPA plan and the Great-West indemnity and preferred provider organization (PPO) plans to provide health insurance for staff. Different contributions to the plans are made by staff on the basis of salary level.

Great-West will not have the 15% increased costs that were projected earlier in the year. The committee recommended changes that reduced the increase to 12% and was happy to report that APA's costs for this health plan will decrease for the 1992-1993 plan year.

2. *Americans With Disabilities Act*. In May the council requested a proposal from APA's legal counsel, Crowell and Moring, for a review of the implications for the Association of the Americans With Disabilities Act.

In September the council reviewed a proposal from legal counsel relative to a training program for management staff and a review of current policies and procedures. This proposal will be reviewed as part of the fine-tuning process for the 1993 budget.

Alan I. Levenson, M.D., chairperson of the *Psychiatrists' Purchasing Group*, talked to the council to respond to questions about the insurance program that were raised during the May 1992 council meeting.

APA-sponsored insurance programs are operated by IPG, a separate corporation. The insurance program structure was established by the Board of Trustees in accordance with federal law.

IPG is trying to educate psychiatrists as much as possible and wel-

comes meeting with any group that wishes to ask questions or provide feedback. APA components can ask IPG to *consider* changing a policy; however, they cannot *direct* IPG to make a particular change, since it is a separate entity.

The *Committee on Advertising*, William D. Strathmann, M.D., chairperson, serves as an advisory body to the editors of APA's three periodicals (*Psychiatric News*, *American Journal of Psychiatry*, and *Hospital and Community Psychiatry* journal) regarding questionable advertisements that have been submitted for publication. The editors ask the committee to review an ad when they cannot come to a consensus, need additional input, or want medical or technical advice. Although the editors usually follow the recommendations of the committee, it is the editors who are legally responsible for the content of their respective publications. The committee has been asked only once this year to review an advertisement.

The council approved revisions of the "Principles and Guidelines of Advertising Acceptance," as developed by the committee. These revisions were intended 1) to expand the guidelines to include tests, devices, and other nondrug entities and 2) to add "country of training" to the list of job-applicant characteristics that cannot be used as the basis for barring applicants from employment. This would represent a new policy decision by APA since it has never before prohibited this kind of discrimination. The guidelines were referred to the Board of Trustees for a final decision.

The Council on Internal Organization welcomes any suggestions or questions from APA members concerning issues that it should discuss in the future.

The Council on International Affairs

Eugene Feigelson, M.D., Chairperson

The Council on International Affairs is the membership component that, in conjunction with components which report to the council, suggests and undertakes activities on behalf of APA that are international in scope. It makes policy recommendations to the APA Board of Trustees when appropriate. It deals with all questions pertaining to psychiatry and international relations that are presented by APA members, other components, psychiatrists from other countries, and staff.

As the membership component most concerned with APA's international activities, the council has become increasingly concerned about the decreasing priority given to these activities in today's rapidly changing world. As other organizations are increasing their international activities, the council and the Office of International Affairs are struggling to address these issues, which are of growing importance in the world today. Our Office of International Affairs has close contacts with over 100 countries and has facilitated contacts for hundreds of APA members for patient referrals and/or collaborative projects. The activities of the council and the committees listed here are of great importance in spreading the values of APA and knowledge gained by American psychiatry throughout the world. There is, in addition, much to be learned from our colleagues in other countries, and collaboration of this sort is of great benefit to American psychiatry as well. We are striving for ways to better inform APA members of the importance of these activities and of the resources already available to them through APA.

The council regrets that the funders of the International Scholars Program at the APA annual meeting have deemed it necessary to eliminate the funds for this program. Not only did this program show that APA has a commitment to facilitating the exposure of our members to international scholars, but it also was a great asset to the scientific

program. Since approximately 10%-15% of all registrants at the annual meeting come from other countries, this meeting has become a truly international one, and we hope to find funding for this important program in the future.

The council had a number of projects of merit this year. In March/April 1992, it organized a bilateral exchange with the psychiatric associations in Poland, Czechoslovakia, and Hungary that took about 13 American psychiatrists to these countries to give lectures and hold discussions on the following topics: schizophrenia and neuroimaging, legal and ethical issues, geriatric psychiatry, psychiatric education, diagnostic issues, consultation-liaison psychiatry, substance abuse, talking therapies, mood and anxiety disorders, psychopharmacology, child psychiatry, and the chronic mentally ill and homelessness. The project was funded by a grant from the Upjohn Company. Approximately 100-125 psychiatrists from each country participated in this exchange. The programs were jointly planned and executed, and reports are available from the APA Office of International Affairs. The APA Board of Trustees has approved the establishment of a corresponding task force for follow-up on this exchange, and the council is hoping to secure funding for the next phase of the project, which would include exchanges with Romania, Bulgaria, Albania, and the Baltic states.

The council has proposed a series of resolutions for the June 1993 World Psychiatric Association (WPA) Congress in Rio de Janeiro. The WPA General Assembly will meet during that time to elect new officers and to vote on any resolutions pending from member organizations. The council has suggested the adoption of an international bill of rights for psychiatric inpatients and a resolution on human rights. In addition, working in conjunction with the APA Committee on Gay, Lesbian, and Bisexual Issues, the council has suggested that APA's

position statement on homosexuality be slightly amended for adoption by the WPA. The council has also suggested the following resolutions for action by the WPA General Assembly:

Resolved, the Executive Committee of the World Psychiatric Association shall explore the feasibility of developing and implementing a set of standards that can be used to assess whether the mental health care systems within the countries of member societies meet a minimum set of requirements that protect the basic rights of the mentally ill and ensure the most appropriate care within the resources available.

and

Resolved, the Executive Committee of the World Psychiatric Association shall develop and distribute educational materials and programs for psychiatrists that can be used to inform them about the basic principles of diagnosis, assessment, and treatment of major mental disorders. Further, that these materials shall be prepared in a manner that is sensitive to the cultures of the countries in which they may also be used by other mental health professional and physicians, especially in developing countries.

Finally, the council has suggested that APA offer to provide in-kind services to the WPA in lieu of dues. The council believes that this would bring more benefit and cost-effectiveness to APA and its members and would give the WPA the benefit of the expertise of our Office of International Affairs.

The council has asked APA staff to look into possibilities for future collaboration with other membership organizations of mental health professionals in the belief that there is much common ground to explore. One step in this direction is the ongoing council contribution to the Sub-Saharan Journal Distribution Program of the American Association for the Advancement of Science.

The *Committee on International Education*, Normund Wong, M.D., chairperson, has received approval of the *International Psychiatric Directory*, which will be published by American Psychiatric Press, Inc., in the spring of 1993. This directory is being compiled and edited by the Office of International Affairs with funding from the Upjohn Company and will provide an excellent resource for individual psychiatrists, medical schools and departments of psychiatry, and libraries. Over 150 countries have been contacted for relevant information. The focus of the directory is on medical and psychiatric education and mental health services.

In addition, the committee is working on a proposal for international standards for psychiatric curricula and is focusing the first phase of its work on South and Central America. Members of the committee have had meetings with colleagues in many of these countries. In December 1992 a meeting was held in Mexico City in conjunction with the InterAmerican Council of Psychiatric Organizations and the University of Mexico; psychiatrists from the United States, Colombia, Argentina, Venezuela, Costa Rica, and Mexico came together to plan a larger conference in Venezuela in 1993. The committee hopes that such a cross-cultural workshop will facilitate an exchange of knowledge and expertise and will further enable the committee to move ahead on similar projects in other parts of the world. Outside funding is being sought for this project.

The *Committee on Human Rights*, Loren Roth, M.D., chairperson, has published the booklet *Human Rights and the American Psychiatric Association*; it can be obtained from the APA Office of International Affairs in single copies, for individuals, or in multiple copies, for organizations. The booklet explains APA activities in the human rights arena—nationally and internationally—and encourages APA members to become involved in this very important field of work. The committee was pleased to give the first APA Human Rights Award to the Committee on Scientific Freedom and Responsibility and the Science and Human Rights Program of the American Association for the

Advancement of Science. The award was presented during the Convocation at the 1992 APA annual meeting in Washington. Recommendations for this annual award should be sent to the APA Office of International Affairs for referral to the committee. The committee organized a Presidential Symposium for Dr. Hartmann during the 1992 annual meeting and was honored by the participation of a number of distinguished specialists in the field, such as Dr. Inge Kemp Genefke of the Rehabilitation Center for Torture Victims in Denmark, Mr. Eric Stover, Executive Director of Physicians for Human Rights, and Dr. Mohamed Mandour of the Egyptian Organization for Human Rights. The committee has organized a workshop for the 1993 annual meeting titled "Pervasive Effects of Political Oppression in Children and Adults."

The committee continues to work on specific case issues in a number of countries throughout the world in response to information received from APA members and from other organizations concerned about human rights. The committee is working with the American Association for the Advancement of Science on an "urgent action" that will, it is hoped, bring more APA members into the human rights arena.

The *Committee on Abuse and Misuse of Psychiatry and Psychiatrists*, Raymond Freebury, M.D., chairperson, has changed its focus from the condemnation of the use of psychiatry for political purposes in specific cases to educational issues related to the rehabilitation of victims and the area of ethics. The committee is also contacting other APA components involved in human rights and ethical issues to ascertain interest in and the feasibility of combining forces in a commission on human rights, which would deal with human rights and psychiatric abuse issues in the United States as well as other countries. It is possible that a proposal on this subject to the APA leadership will be forthcoming. The committee has made the following statements in this regard:

As APA members, we deplore the abuse of psychiatry within the United States, and we believe that the APA has the obligation to do everything possible to defend the rights of patients to non-abusive and ethical treatment.

If this committee is to have maximum credibility in criticizing the abuse of psychiatry abroad, it needs to be equally vigorous, or at least collaborate with equally vigorous collegial APA committees, in assuring that abusive or unethical psychiatry in the United States receives the same degree of scrutiny as does such psychiatry abroad.

Since human rights is a universal issue, not merely one which applies to foreign countries, the committee felt that this discussion lent further impetus to pursuing the idea of combining forces with other APA components in a reorganized structure.

In the meantime, the committee works on cases of abuse and neglect in a number of different countries, including Cuba, Mexico, Romania, and countries of the former Soviet Union. In addition, the committee continues to explore development of a plan for coordinating the distribution of donated educational materials to the independent psychiatric associations of the new republics of the Commonwealth of Independent States. The committee is exploring the translation into Russian of an appropriate current handbook of psychiatric treatment for distribution to these associations.

The council and components plan to consider their advocacy role for APA's international activities and appreciate the interest of the membership. Many psychiatrists all around the world look to APA for collaboration, and the council believes that we have an obligation to strengthen these activities. There is much to be learned from these colleagues, and APA cannot afford to take a totally national approach to our profession. Increased communication and collaboration can only strengthen the profession nationally and internationally, and the council will continue to fight for this approach.

The Council on Medical Education and Career Development

James Shore, M.D., Chairperson

The council has overall responsibility for all issues and efforts related to medical education, for both psychiatrists and, where appropriate, other physicians. Its responsibility includes promoting quality education and training for medical students and residents, as well as continuing education activities for practitioners. It is involved with activities related to subspecialties and special areas of clinical interest, such as child and adolescent psychiatry, forensic and consultation-liaison psychiatry, and administrative psychiatry. In addition, the council has primary responsibility for implementing and monitoring the APA continuing medical education requirement for the membership, in order to promote the highest quality of psychiatric care and to encourage continued professional growth of psychiatrists.

In collaboration with its 18 components, other APA components, and related psychiatric, medical, and educational organizations, the council continues to be active in the recruitment of medical students into psychiatry. The decline in psychiatric recruitment is well documented from 1988, when 745 U.S. graduates entered psychiatry residency training through the National Resident Matching Program, to 526 in 1992. In response to these statistics, the council designed a 12-step action plan that clearly makes recruitment the top priority. The initiatives to support recruitment into psychiatry are the following.

1. *To stimulate studies that will broaden understanding of medical student recruitment trends.* At present, recruitment planning is less effective because of an inadequate data base. For example, it will be valuable to develop recruitment profiles for each medical school and to identify the important variables associated with high and low recruitment experiences.

2. *To design a national site visitor/consultant project, focusing on the enhancement of medical student recruitment and medical student education in psychiatry.* For over a decade, psychiatric education significantly benefited from the site visitor program of the National Institute of Mental Health (NIMH). During that era, departments maintained a sharper focus on programs for medical student education and were consistently reminded by the site visitors of the mechanisms essential to enhancing medical student recruitment. The possibility of reviving a national site visitor/consultant project is being carefully evaluated.

3. *To stimulate critical liaisons with other key psychiatric organizations.* A successful recruitment agenda requires that all major psychiatric organizations address recruitment simultaneously, developing an effective conjoint effort. The Association of Directors of Medical Student Education in Psychiatry, the American Association of Chairmen of Departments of Psychiatry, and the American Association of Directors of Psychiatric Residency Training, in addition to subspecialty societies, all must join with APA in a coordinated recruitment initiative.

4. *To emphasize the APA liaison with the Association of Directors of Medical Student Education in Psychiatry.* This organization is critical to the success of the APA recruitment initiatives. These directors have the greatest access to and primary influence on the potential pool of medical student applicants. It is the intent of the council to work actively in the support of the Association of Directors of Medical Student Education in Psychiatry and to broaden its relationship with APA.

5. *To enhance the role of district branches in recruitment.* The district branch leadership and membership represent a critical resource for the recruitment of medical students into psychiatry. The council will identify mechanisms and encourage district branch participation with departments of psychiatry and medical student classes.

6. *To establish active collaboration among the council, its components, and the Ethics Committee for development of curricular materials.* Recent publicity has distorted psychiatry's image and reinforced a negative reputation, which may have contributed to the recruitment

decline. It is essential that APA components collaborate on and work for model educational programs that include appropriate ethical issues in the guidelines.

7. *To ensure wide distribution of recruitment videotape materials.* Under the sponsorship of the council and the Committee on Medical Student Education, copies of the videotapes on three major psychiatric conditions were distributed to all departments of psychiatry. The conditions are panic disorders, anxiety disorders, and depression, and the tapes illustrate a variety of patients being treated with different modalities by psychiatrists. The council believes that the videotapes and the accompanying educational material will be useful both in the recruitment of medical students into psychiatry and in increasing the awareness of all students.

8. *To review curricular guidelines.* The curricular guidelines published a decade ago will be reviewed and updated to present a model for the field.

9. *To develop a handbook for use by directors of medical student education.* The Office of Education will evaluate the feasibility of developing a new handbook for directors of medical student education to be used as a resource document, directed especially at all new directors of medical student education.

10. *To review and enhance the role of residents in recruitment of medical students.* Psychiatric residents often are the most valuable teachers and recruiters. The Committee on Graduate Education and the Committee of Residents and Fellows will review residents' role in student recruitment and encourage this critical involvement. These committees will revise the pamphlet "Psychiatric Resident as Teacher," which provides guidance for residents in their important role as teachers to all medical students. This will facilitate their special role as recruiters.

11. *To revise the format of the Directory of Psychiatry Residency Training Programs.* This directory has served as a critical resource in the field for all medical students with an interest in psychiatry. If students decide to pursue a psychiatric career, referring to the directory is the first important step in their selection of a training program.

12. *To support the five recommendations from the May Conference on Recruitment sponsored by the American Association of Directors of Psychiatric Residency Training.* The following five recommendations were specifically directed at chairpersons and at departments of psychiatry:

- a. To identify psychiatrists who are directors of medical student education and have adequate resources.
- b. To develop a specific recruitment program in psychiatry; a faculty member must be identified to coordinate this process.
- c. To identify students with an interest in psychiatry.
- d. To identify special educational programs to maintain and foster an interest in psychiatric careers.
- e. To develop special programs to focus faculty attention on psychiatric education and recruitment.

The *Work Group on Accreditation and Certification*, Jerald Kay, M.D., chairperson, was established in 1991 after a 1-day meeting of two representatives each of APA, the American Board of Psychiatry and Neurology (ABPN), and the Residency Review Committee for Psychiatry. The council convened the meeting to address issues related to accreditation of subspecialty fellowship training programs and certification of individuals completing such programs. It was the consensus of the group that the meeting was not only productive but an appropriate first step in addressing the broad issues related to subspecialty training. It was agreed that continued dialogue was needed to address these issues fully, and the council recommended and the Board of Trustees approved formalization of the Work Group on Accreditation and Certification to enable it to continue this work and to collaborate with other organizations (such as the American Association of Directors of Psychiatric Residency Training, American As-

sociation of Chairmen of Departments of Psychiatry). Other recommendations included the following.

1. Modification of the charges for the Committee on Graduate Education to reflect responsibility in the areas of postresidency education and training, including responsibility and collaboration in the review and assessment of requests for added qualifications in subspecialties.

2. Further discussion directed at developing APA positions on subspecialization and related training; the work group should draft a position paper that reflects the history of and current issues in the subspecialization process.

3. A letter from the Medical Director to both the American Association of Directors of Psychiatric Residency Training and the American Association of Chairmen of Departments of Psychiatry requesting their views on accreditation and certification of postresidency experiences.

4. A letter from Dr. Kay to all involved psychiatric and educational groups soliciting feedback regarding research requirements in fellowship training, the appropriate length of fellowships, and other issues relevant to subspecialty fellowship training.

The *Committee on Administrative Psychiatry*, Michael Vergare, M.D., chairperson, reported the results of the oral portion of the examination for certification in administrative psychiatry held on May 1, 1992, during the APA annual meeting in Washington, D.C. In September the committee reviewed the criteria for acceptance for examination, with an emphasis on recognizing the changing roles that psychiatrists play in administration.

Dialogue with the Committee of Residents and Fellows and the Committee of Early Career Psychiatrists included discussion of future emphasis on education, as well as certification in administrative psychiatry. Course work in administrative psychiatry during residency training and for continuing education programs will be explored further.

Donald Langsley, M.D., was named the recipient of the 1993 Administrative Psychiatry Award.

The *Committee on Graduate Education*, Daniel K. Winstead, M.D., chairperson, focused much of its energy on issues related to recruitment of medical students into psychiatry. The committee reviewed the 12-step program developed by the council and endorsed this document as an important initial approach to this critical problem. The committee also felt that in the long run it would be useful for psychiatrists to be more involved in undergraduate education and in the medical school admissions process, in order to ensure that more students enter medical school with some knowledge of and potential interest in psychiatry. The issue of adequate remuneration for psychiatry residents and fellows, as well as the key role that residents and fellows play, must be addressed in the face of increased indebtedness by medical school graduates. The committee acknowledges the role that residents play in teaching medical students and in the recruitment process, and it suggested slight modifications to the brochure "Psychiatric Residents as Teachers," along with wider distribution of this helpful document. The committee also recommended that the *Directory of Psychiatry Residency Training Programs* be prepared in computer disk format to allow for easy retrieval and that one of these disks be provided to each medical school at a reasonable cost. An informational brochure on psychiatry should be prepared for distribution to interested medical students, residents, and others, advising them of the availability and use of the directory.

Issues related to subspecialization were also considered in great detail. The committee reviewed the draft special requirements for added qualifications in geriatric psychiatry that have been prepared by the Residency Review Committee for Psychiatry. In general, these requirements were quite useful, and the core requirement for faculty and trainees is essential for ensuring quality. The committee recommended that these requirements be modified and contain new wording to ensure that programs have didactic and clinical experiences in psychotherapy and in the emergency management of the older patient. In addition, it felt that these requirements should more clearly state that the purpose of these training programs is to train individuals for leadership roles in order to advance the field of geriatric psychiatry.

On a more controversial note, the committee carefully reviewed the proposal for added qualifications in consultation-liaison psychiatry and ultimately decided that it disagreed with the Commission on Subspecialization in that this proposal failed to meet several of the key

criteria necessary for approval of added qualifications. The proposal, if accepted, would have a negative impact on the training of general psychiatrists by moving requirements for skills in consultation-liaison psychiatry to the fellowship level. Furthermore, this would set a dangerous precedent by further fragmentation of general psychiatry.

During a joint meeting with the Committee of Residents and Fellows, discussion focused on a position paper on moonlighting prepared by Dr. Jules Bemporad. The committee also reviewed the 1969 APA position statement on moonlighting, particularly in light of recent regulations and policy decisions regarding resident work hours. The committee urges that APA discourage blanket prohibition of moonlighting and allow individual programs to decide this issue.

The *Committee on Consultation-Liaison Psychiatry and Primary Care Education*, Fawzy I. Fawzy, M.D., chairperson, reported on the establishment of the *Subcommittee on the Psychiatric Aspects of Life-Sustaining Technology*, chaired by Maurice Steinberg, M.D. This subcommittee was charged to explore psychiatry's role in the area of life-sustaining technology.

A workshop jointly sponsored by the committee and the Society of General Internal Medicine was presented at that organization's annual meeting. "Making Sense of Personality Disorders: A Psychodynamic Approach for the Internists" focused on the development of teaching workshops and educational programs for internists. The committee will be submitting a proposal for a follow-up workshop at the next annual meeting of the Society of General Internal Medicine.

The committee endorsed the proposal to undertake a survey of residency programs to determine the range of consultation-liaison teaching time among residencies.

Work with Dr. Harold Pincus, Director of the APA Office of Research, continues on the proposed diagnosis of "minor anxiety/depression" for DSM-IV; the primary concern is the political issues involved.

The *Committee on Continuing Education*, Pauline Langsley, M.D., chairperson, is working to ensure that APA meets all standards of the Accreditation Council for Continuing Medical Education when it is surveyed for reaccreditation in 1994. As a result, the committee devised a procedure whereby APA would be able to continue to jointly sponsor continuing education activities of district branches while still being involved in program planning and evaluation for jointly sponsored programs to the extent required by the Accreditation Council for Continuing Medical Education.

The committee developed a mission statement for APA, as required by the accreditation council, and is also involved in developing a disclosure policy and form for faculty of educational programs, which requires identification of any financial relationships faculty members have with manufacturers of products that will be discussed in the faculty members' educational sessions.

The major thrust of the *Committee on the Impaired Physician*, Gordon Moore, M.D., chairperson, has been to explore ways that psychiatric understanding can be more effectively used to address problems in this area. To a large extent, this requires educating people and professional bodies who make judgments regarding physician behavior. Specifically, it is important to review the balance between evaluating impaired physicians for chemical dependency and/or psychiatric disorder, the boundaries between ethics and impairments, and contributions of psychiatrists in this process. To meet these goals, committee members are individually involved with physician impairment programs and make presentations at appropriate meetings and workshops. The committee completed a set of guidelines, "Mental Health Services for Medical Students and Residents," and is developing a videotape on physician impairment for distribution to medical schools for the purpose of educating medical students and residents. There continues to be concern about potential discrimination against a once-impaired physician (such as denial of insurance or employment), the method of psychiatric input to physician impairment committees, and development of explicit criteria for impairment.

The *Committee on Medical Student Education*, Carol A. Bernstein, M.D., chairperson, reported on its efforts over the past year to address the decrease in the number of medical students choosing careers in psychiatry and ways to enhance recruitment activities. Some of the committee's recruitment initiatives have included the following.

1. Distribution of the videotape series on psychiatric disorders (developed by the Division of Public Affairs) to every U.S. medical school,

along with the recommendation that the videotapes be used as a "springboard" for a recruitment night with residents and faculty to discuss career opportunities in psychiatry. The committee will conduct a follow-up study to determine the effectiveness of these videotapes as recruitment tools.

2. A telephone poll of 35 medical schools that were successful in recruiting medical students into psychiatry from 1983 to 1990. Results of the poll will be distributed to all directors of medical student education in psychiatry and directors of residency training throughout the country.

The committee sponsored a highly successful workshop on choosing a psychiatry residency program for medical students at the 1992 annual meeting in Washington, D.C.. This workshop will be repeated in 1993. "Medical Student Day" and the annual luncheon for medical students, residents, and training directors continue to be enormously successful. Approximately 250 medical students, residents, and faculty attended this year's luncheon, at which the 58 recipients of the 1992 Nancy C.A. Roeske, M.D., certificate were honored. There are 60 nominations for the 1993 award.

Other important activities of the committee include the following.

1. Plans to update "Curriculum Guidelines in Psychiatry" and the "Choosing Psychiatry" brochure, in collaboration with the Association of Directors of Medical Student Education in Psychiatry.

2. Revision of the exhibit structure used by the Office of Education at the annual conventions of the American Medical Student Association and the Student National Medical Association, to be available for the 1993 meetings.

3. Development of ways to encourage medical student membership in APA, for example, recommending "adopt a student" programs to departments of psychiatry and district branches throughout the country.

4. Work with the Local Arrangements Committee and the Office of Education to publicize the 1993 annual meeting to medical schools in the San Francisco area, including plans to hold an orientation program for medical students on the first day of the meeting.

5. Continued collaboration with allied organizations, such as the Association of Directors of Medical Student Education in Psychiatry, American Association of Directors of Psychiatric Residency Training, and the Association for Academic Psychiatry.

The *Committee of Residents and Fellows*, Ivan C.A. Walks, M.D., chairperson, continues to address the educational aspects of a wide range of issues affecting psychiatry and psychiatric education and training. These issues include trainee indebtedness, recruitment of medical students, the economics of psychiatric practice, and the changing roles of the psychiatrist in both mental health and general medical care. To this end, the committee has expanded its communication and collaboration with other APA components.

The destigmatization and demystification of psychiatry are areas of particular focus. The Committee of Residents and Fellows is working to formalize the psychiatric trainees' role to increase recruitment of medical students into the field. In addition, the committee will work with the Assembly Committee of Area Member-in-Training Representatives and Deputy Representatives to explore the possibility of holding sessions for medical students at the district branch level, similar to the "Meet the Experts" session held at each annual meeting.

Expanding the interface of psychiatry with public and political concerns continues to be a priority. To this end, the Committee of Residents and Fellows reaffirms its interest in formal ethics instruction, in general, and, specifically, in managed care, reimbursement, and access to care. Recognizing the importance of trainee involvement in organized psychiatry, the committee will work with the Office of Education to explore a local resident network to assist with housing accommodations for trainees attending APA annual meetings. At the 1993 annual meeting the Committee of Residents and Fellows will sponsor a workshop on moonlighting. Further, it will continue discussions with the Committee on Graduate Education to emphasize that moonlighting is a necessity for many residents and to discourage a blanket prohibition.

The committee continues to write and publish the *Psychiatric Residents' Newsletter*, which now includes a special column for medical students in each issue, and to serve on the editorial board of the *Jefferson Journal of Psychiatry*.

The *Task Force on Diagnostic Education*, Michael A. Fauman,

Ph.D., M.D., chairperson, has proposed an educational program for the introduction of DSM-IV that includes the following components: 1) identification of key diagnostic points that highlight important conceptual elements essential in differential diagnosis; 2) development of a library of brief diagnostic video "trigger" vignettes of real and simulated patients that highlight difficult areas of differential diagnosis, especially those associated with key diagnostic points; 3) presentation of annual meeting symposia and workshops, administered through the Office of Education, that focus on the diagnostic process in psychiatry and the use of DSM-IV. The task force is also working with the Task Force on DSM-IV to present joint programs at the annual meeting. The main goal is to develop educational tools and activities that will help clinicians understand DSM-IV and apply it consistently to patients in the real world.

The *Task Force to Facilitate Communication Between APA and ABPN*, Rodrigo Munoz, M.D., chairperson, has focused on increasing the number of minority directors on the ABPN board. So far, the efforts have not been successful, in part because of the minimal representation of minorities among the senior examiners and the small proportion of minorities among other examiners and consultants. The task force has continued conversations with representatives of the ABPN in an attempt to obtain a higher participation of minorities (especially international medical graduates) among the examiners, consultants, and ABPN directors.

With the help of the council and in cooperation with John Oldham, M.D., chairperson of the Scientific Program Committee, the task force is proposing APA-sponsored courses at the annual meeting to help ABPN candidates. The same courses could be provided by the district branches.

The task force will invite a representative from the Mexican Psychiatric Association to discuss various ways to obtain better interaction with APA and the ABPN. Interest in this kind of interaction has increased because of the recently signed North American Free Trade Accord.

The *Work Group on Recertification*, Gordon Strauss, M.D., chairperson, has been meeting and working with the ABPN recertification committee to exchange ideas about plans for recertification. There is general agreement that APA should share with the ABPN the overall responsibility for the recertification process: the ABPN would be responsible for the actual recertification test materials, and APA would focus on the educational aspects of recertification preparation.

The *Committee of Early Career Psychiatrists* (formerly the Committee of Young Psychiatrists), Mary Ellen Foti, M.D., chairperson, has completed a demographic survey of early career psychiatrists in conjunction with the Office of Membership. Nearly 16.6% of APA members are early career psychiatrists (under 40 years of age or within 5 years of residency/fellowship training). Other points of relevance are 1) that a higher percentage of early career psychiatrists are female (38.8%) than male (22.7%) and 2) that early career psychiatrists are as likely as other general members to be board certified (67%). The committee will continue to work with the Office of Membership to further define this segment of the general membership.

The committee has developed a bibliography of useful articles pertinent to aspects of establishing a private practice, e.g., forensic issues. It is working with the APA Lifers group to develop a mentorship program. Information regarding these projects will be available during the 1993 annual meeting at the Office of Education exhibit in the APA Resource Center.

Through the efforts of Drs. H. Paul Putman III (delegate) and James C.Y. Chou (alternate delegate), the committee remains active in the Young Physicians' Section of the American Medical Association.

The *APA/Mead Johnson Fellowship Selection Committee*, Stuart Keill, M.D., chairperson, selected the 1992-1993 APA/Mead Johnson Fellows during its June 1992 meeting. There were no major discrepancies in the ranking among the members of the committee. The committee received 41 nominations, from which 15 fellows were selected.

Because of budgetary constraints, the fellowship benefits have been reduced through a limitation on the number of fellows whose attendance at the APA annual meeting will be financially supported. During 1992-1993 up to three fellows will receive funds to support their attendance at the meeting.

The remainder of the fellowship benefit is unchanged. All 15 fellows will receive financial support to attend and participate in the

1992 and 1993 Institutes on Hospital and Community Psychiatry. In addition, five residents who are selected by the fellows themselves will serve as liaisons to a variety of APA components and to the American Association of Community Psychiatry.

The *APA/NIMH Minority Fellowship Selection and Program Committee*, Charles Pinderhughes, M.D., chairperson, reviewed 21 applications for the 1992–1993 APA/NIMH Minority Fellowship Program. Eight residents were accepted: one Asian man, one Asian woman, two Hispanic men, two black men, and two black women. In addition, the committee named five 1991–1992 fellows as trainee-consultants (“trainee-consultant” is an honorific designation, and funding for participation is not provided). Dr. Melvin Sabshin will serve as interim director of the fellowship program until a successor to Dr. Jeanne Spurlock is named.

The committee is conducting a survey of 224 directors of psychiatry residency training programs to identify resources and needs for cross-cultural material in psychiatry training programs around the country. The data collected will be used to make recommendations to the Council on Medical Education and Career Development on a variety of points related to cross-cultural issues in training. Dr. F.M. Baker, a former committee member and author of the survey questionnaire, serves as an advisor to the committee.

The *APA/Burroughs Wellcome Fellowship Selection and Program Committee*, Roger Peele, M.D., chairperson, reviewed 42 nominees for the 1992–1994 fellowship program and selected 10 new fellows. Following the 1992 annual meeting, staff in the Office of Minority/National Affairs worked on the liaison assignments of the new fellows, which were based on feedback from the fellows and component chairpersons. (Whenever possible, fellows were assigned to one of their five choices.)

On behalf of the Burroughs Wellcome Company, Mr. Jerald Breitman presented a check for \$115,000 to the Board of Trustees in May

to support the fellowship program. A reception was also hosted by Burroughs Wellcome for current and former fellows, representatives of the Maurice Falk Medical Fund, and members and staff of APA who have been involved with the program.

The APA/Burroughs Wellcome Fellows workshop at the 1992 annual meeting, “Skeletons in Our Closet: Suicide, Drugs and Psychotherapy,” proved to be highly successful: Dr. Paul Fink was the discussant. In addition, the fellows and other APA resident groups sponsored the forum “How and Why to Lobby on Capitol Hill.”

The research symposium, held in September, featured the presentations “Race and Involuntary Commitment in Rural South Carolina” (Stephen McLeod Bryant, M.D., Assistant Professor, Department of Psychiatry and Behavioral Sciences, Medical University of South Carolina) and “Research Opportunities for Psychiatrists at NIMH” (Frederick Goodwin, M.D., Director of NIMH). In addition, a current APA/Burroughs Wellcome Fellow (David B. Robinson, M.D., M.P.H., Fellow, Division of Child and Adolescent Psychiatry, University of North Carolina School of Medicine) presented “Correlating Influences of Career Focus, Personal, and Didactic Factors on Resident Entry Into Personal Psychotherapy: A Biological vs. Psychodynamic Split?”

The fellows exchanged ideas and expressed their enthusiasm about being involved in the APA governance process. They have made excellent use of the unusual opportunity to participate in the work of APA components, including the Board of Trustees and the Joint Reference Committee, and emphasized the need for continued participation in this process, on both the national and local levels, in order to retain and use the skills acquired during the fellowship.

The APA/Burroughs Wellcome Fellowship newsletter is published three times a year, and Falk and APA/Burroughs Wellcome Fellows (current and alumni) are encouraged to submit articles pertinent to resident issues and concerns.

The Council on National Affairs

Fred Gottlieb, M.D., Chairperson

APA's charge to this council is to bear responsibility for matters of national interest to psychiatry and the patients it serves, with particular attention to those social and health-related issues that affect underrepresented and underserved groups in the general population and within the profession. The charge notes that the multiple approaches to such problems should include emphases upon clinical care and research, service delivery and systems of care, and all forms of prevention.

To try to deal comprehensively with such issues would necessitate using all the resources of APA. Fortunately, over the years the council has recognized the complementarity of other councils' tasks. So it has narrowed its focus in two ways: 1) to function especially in regard to the social and health-related issues of the underrepresented and underserved and 2) to serve as an organizing nidus for underemphasized but very significant psychiatric issues within our society. It is not inadvertent that the Task Force on Psychiatric Dimensions of Disasters is “housed” within the council, as is the Committee on Psychological Aspects of Nuclear Issues; similarly, the Committee on Religion and Psychiatry and the Committee on Abuse and Misuse of Psychiatry in the U.S., viewed by a few as somehow tangential, are also decidedly salient aspects of our own, and every society's, apt tension in determining its values, scientific and humane.

Significantly, this year the council offered to house an APA committee component on the homeless, when the Council on Psychiatric Services' current Task Force on the Homeless is terminated. Clearly, such

an APA component would address issues of extraordinary national scope, well fitting the council's mandate. The Council on Psychiatric Services recommended forming such a committee and placing it in the Council on National Affairs. In December 1992 the Board of Trustees gave its approval for this committee, subject to budget allocations.

Although the council's closest linkages this year were with the Council on Children, Adolescents, and Their Families, the Council on Psychiatric Services, and the Assembly Committee on Minority/Underrepresented Groups, in fact the council necessarily regularly works very closely with representatives from the Assembly, staff, and a great many other components. Besides the components noted in paragraph two of this report, others that specifically report to the council include the seven nominally focused on so-called “minority” issues: the Committees of/on American Indian, Alaskan Native, and Native Hawaiian Psychiatrists; Asian-American Psychiatrists; Black Psychiatrists; Hispanic Psychiatrists; International Medical Graduates; Gay, Lesbian, and Bisexual Issues; and Women. It should be obvious that our organization, as well as our society, is and must continue to recognize itself as a “majority of minorities.”

When the council met in February 1992, it discussed possible ways to improve communication between itself and its components and between the components themselves. As agreed then, the council members subsequently surveyed the component chairpersons for suggestions. Among many others, those included the following: 1) an early working meeting of component chairpersons at the fall compo-

nent meetings, who thus could, in advance, be aware of each others' agendas and become more aware of both the communality of their activities and also the potential for conflictual areas to be resolved; 2) preferences regarding ongoing rather than rotating component liaisons from among members of the council; and 3) the possibility of a specific orientation in which all new members of the Council on National Affairs' components would have a chance to meet and talk with each other. The council discussed ways to implement these suggestions promptly. In addition, the council emphasized the utility of component chairpersons contacting each other about areas of common interest or concern. The council similarly encouraged its components to freely contact other components of the Association, with no a priori requirement to go through the council beforehand, but with the hope that the council would be helpful for consultation or advice about such linkages.

A clear example of problematic communication, and serious conflict, occurred when the initial recommendation for recipient of the Solomon Carter Fuller Award by the Committee of Black Psychiatrists was a person who was actively involved in activities contrary to stated APA positions. Many hours of intense discussion occurred during the May and September council meetings, as well as during the intervening months. And questions about a potential Simon Bolivar Award recipient served to underscore the need for a well-delineated functional way to obtain wide input about potential awardees, the scope and meaning of the awards within the context of APA, and the authority to determine the recipient. The council drafted material for the Committee on Grants and Awards to study, insofar as it has been asked to make recommendations to the Board on these matters. The council was very pleased to support unanimously its committees' recommendations of child advocate attorney Marian Wright Edelman for the Solomon Carter Fuller Award and psychiatrists A. Anthony Arce and Paul R. Fleischman for the Simon Bolivar and Oskar Pfister Awards, respectively.

Also at the February 1992 meeting, the council noted that our society expresses concern about violence, often episodically, but that psychiatric activity in this area seems to have been quite circumscribed. In further exploration, it turns out there is considerable knowledge about this endemic major problem and there has been some significant work by psychiatric professionals. Members of some of those groups whose welfare this council is particularly charged to consider often find themselves bearing specific witness to nearly routinized tragedies of violence in their daily lives. While that emphasizes some especially vulnerable sectors of our population, the reality is that the problems of violence affect the whole of our society's citizenry and the structure of the society as well. We felt that this council's diverse components provide the potential for a coalition that, if united, might be able to synthesize innovative contributions to the solutions of such problems and also could serve to activate other APA components.

With the foregoing in mind, the council organized a workshop on violence during the September components meeting. There were brief presentations by distinguished resource persons, who then answered questions and helped to focus discussion. The resource persons included Dr. Sandra Kaplan, chairperson of APA's Committee on Family Violence and Sexual Abuse; Dr. Robert Phillips, chairperson of APA's Committee of Black Psychiatrists; Dr. Terry Stein, vice-chairperson of the Council on National Affairs; and Dr. Mark Rosenberg, Acting Associate Director for Public Health Practice, U.S. Centers for Disease Control. Despite jet lag and, for many, a full day of meetings, the approximately 70 attendees stayed until almost 11:00 p.m. Many thanked the council for organizing the workshop and for providing a unique opportunity to exchange views. On the basis of the feedback from the participants, the council submitted a proposal for a derivative symposium for the annual meeting, to be chaired by Dr. Terry Stein and adding Dr. Carl Bell and Dr. Gail Robinson as participants. We are pleased that the Scientific Program Committee has accepted this proposal for presentation in May 1993. But the council knows, and reminds the membership, that talk alone is no substitute for effective social action.

The council also anticipates that the symposium format will be repeated at the fall 1993 meetings, to address the topic of racism.

After council discussion about a position statement on bias-related incidents, Drs. Terry Stein and Mchecko Graves worked on the

text, copies of which were sent to the council and distributed to component chairpersons. The council unanimously approved the following position statement, which was submitted to the Joint Reference Committee:

Bias-related incidents arising from racism, sexism, intolerance based on religion, ethnicity, and national/tribal origin, and anti-gay and lesbian bias are widespread in society and continue to be a source of social disruption and individual suffering and trauma. These incidents are ubiquitous and occur in both urban and rural areas. Such hate-based incidents consist of acts of violence, abuse or harassment and result in emotional and physical trauma for individuals, as well as stigmatization of affected groups. These ethnic and cultural biases serve to frustrate the basic human need for a sense of dignity, resulting in a despair and hopelessness among the victims that ultimately affect the nation as a whole.

APA has gone on record as deploring racism and other forms of social prejudice, e.g., Position Statement on Homosexuality and Civil Rights, April 1974; United Nations Draft Program for a Decade of Action to Combat Racism and Racial Discrimination, June 1974; Position Statement on Affirmative Action, June 1978; Resolution Against Apartheid, January 1985; Report of the Task Force on Human Rights, November 1985; APA Statement Regarding South Africa, June 1986.

Building upon this tradition, APA now encourages its component groups, including its District Branches, to respond actively to local bias-related incidents through such efforts as: 1) offering leadership in promoting conflict resolution; 2) providing education related to racism, sexism, violence, poverty, homophobia, and other social problems that contribute to such incidents; and 3) establishing prevention programs. APA's Office of Minority/National Affairs is responsible for overseeing and assisting in the implementation of responses that may occur at local, state, and regional and national levels.

Interventions such as those that have occurred in New York City positively involving the psychiatric profession and the local community in response to recent racial incidents can serve as examples of the type of expertise that psychiatrists provide. Using the biopsychosocial model to understand and respond to racism, prejudice and discrimination, psychiatrists can assist their communities in establishing therapeutic environments to begin the healing process, efforts which have already been started in many cities around the country. APA urges its members and component groups to undertake such programs to assist with the resolution of one of America's most challenging issues.

At its October meeting, the Joint Reference Committee declined to recommend the material as an APA position paper in the form presented here, suggesting correctly that it provided both a position statement and a "call to action," which would be better utilized if differentiated. The material has been rewritten and submitted in different forms to the Assembly, which affirmed both theoretical and action aspects at its fall 1992 meeting. The Board approved the position statement on bias-related incidents in December 1992.

Way back in 1987, the Assembly passed an action paper calling for the development of cross-cultural curricula to be used by the Residency Review Committee for Psychiatry in evaluating residency training programs for accreditation, urging that such work be accomplished with participation by the Committee on Minority/Underrepresented Groups in the Assembly. The proposal was then approved at appropriate APA levels. But trying to elicit and then coalesce six separate curricula, done by different groups and individuals, resulted in the passage of a lot of time.

The council previously reported to the Joint Reference Committee that the council wished to bring this matter to a successful conclusion, inasmuch as it began to seem unlikely that we could even obtain all the curricula, let alone make them consistent with each other. The council is pleased that we have now been able to obtain drafts, which were reviewed by the council members between the components' meetings and the Joint Reference Committee meeting. The reviews were enthusiastic about the riches, importance, and potential useful-

ness of the curricula. Concurrently, the reviews each suggest problems in one or more of the curricula and suggest the desirability of added standardization, as well as a clear introduction and rationale if they are to be presented as a coherent unit. Moreover, realistic questions were raised about the inclusivity and demandingness of such curricula if they were adopted by a training program in their entirety. When viewed as a unit they seemed more analogous to a buffet table than to an organized, sequential meal.

To expedite the publication of these curricula, the council suggested that the APA Office of Education quickly participate in this project, at least informally. Moreover, if it appears that reasonably prompt publication as an organized whole cannot be accomplished, because the council is very concerned about further lengthening what has already been a very long time since the project was authorized, we believe the material may need to be released to the initial writers for separate submissions. We felt, if need be, the curricula could be printed individually, serially or not, in one or more APA documents or published by American Psychiatric Press or submitted to appropriate journals, e.g., *Journal of Academic Psychiatry*. By the time of the council's February 1993 meeting, the council anticipates the situation will have clarified enough that one of those options can be chosen.

When the council met with the Joint Commission on Government Relations, the topics discussed included how to better develop processes that are more than merely reactive to health care reform proposals, inquiry about if and how APA's Committee on Universal Access to Health Care actually is utilized by the commission, and concern regarding setting priorities, e.g., the Division of Government Relations' activity level in the anticipated veto of family leave legislation. With regard to the latter, the council attempted to challenge the Joint Commission on Government Relations in its apparently routine use of the rationale that we need to use our "chits" in the legislative game very sensibly. That might *not* always be the way to proceed. Sometimes, even when we think it is an issue on which we think we are going to lose, perhaps it is still appropriate to go all out, just because it is "right."

Drs. Terry Stein and Nada Stotland represented the council at a meeting with the Joint Commission on Public Affairs on Thursday afternoon of the fall components meetings. The commission was interested in the issue of violence, particularly domestic violence, and wanted to know APA's position with regard to the position of the American Medical Association (AMA) on domestic violence. As is the case with a number of other APA position papers, it needs to be reviewed for germaneness and timeliness, in view of changing social currents, scientific advances and technological shifts, and political significance.

The council also invited Medical Director Dr. Melvin Sabshin to meet with it to discuss the status of recruitment for the persisting vacancy in the post of Deputy Medical Director for the Office of Minority and National Affairs, for which a search committee has been presumed to have been active during most of 1992. Our presumption was wrong. Dr. Sabshin disclosed his preliminary thoughts about reorganization, e.g., the possibility of combining certain of the functions hitherto in the Office of Psychiatric Services with the Office of Minority and National Affairs, as well as changing the job description for the search committee's explorations. Time precluded extensive discussion, but the council subsequently voiced considerable caution about the potential change, expressing hope that additional exploration and wider input might result in the emergence of other possibilities. This promises to be a significant agenda item for the winter 1993 council meeting.

Following are reports on the 1992 activities of the components that report to the Council on National Affairs.

The *Committee on Abuse and Misuse of Psychiatry in the U.S.*, Ledro R. Justice, M.D., chairperson, received many helpful comments on its draft paper "Misuse and Abuse of Psychiatry" after the 1991 fall component meetings. A carefully revised definition emerged, i.e., "Abuse of psychiatry refers to the mistreatment, injury or abuse of patients which may result when the psychiatrist's role is misused; that is, used absent the requisite, normative, ethical emphasis on the patient's well-being." Other revisions are pending, with Dr. Jeremy Nahum serving as editor, Drs. Humberto Martinez and William Womack working on the special issues section, and Dr. Justice and Dr. Harry Prosen working on the institutional section.

Mindful of the need for an APA statement on abuse and misuse of psychiatry, especially in light of recent events, the committee drafted such a statement, which the Council on National Affairs approved for submission to the Joint Reference Committee. It is remarkably terse and seemed unlikely to elicit objection or resistance, reading as follows:

The American Psychiatric Association condemns the use of psychiatric knowledge, practice, and institutions for reasons other than ethical evaluation and treatment of patients, research, and education. When used to further organizational, political, social, or financial objectives to the detriment of competent and ethical care, then it is an abuse or misuse of psychiatry.

To the council's surprise, the Joint Reference Committee returned the draft position statement, asking that additional context be provided and suggesting that the phrase "antithetical to" be considered as a substitute for "other than" in the first sentence of the statement. The committee will recommend appropriate changes to the council such that a needed position paper can be resubmitted this spring to the Joint Reference Committee, Board of Trustees, and Assembly for their potential approval.

Several allegations of abuse and misuse of psychiatry were forwarded to the committee for its review. After studying and discussing these allegations, the committee concluded that it was not within its purview to address them and will so inform the parties concerned.

The *Committee on American Indian, Alaska Native, and Native Hawaiian Psychiatrists*, Albert Samuelson, M.D., chairperson, was part of a team assembled by APA's Division of Government Relations, which included government representation, for a highly successful site visit to Indian reservations near Billings, Mont. APA's Division of Government Relations is reviewing the committee's report. It is more and more evident that relevant APA components should be dealing directly with issues affecting the mental health of American Indians. The committee will try to arrange informal liaison between itself and other pertinent components.

In September the committee visited Mr. Steven Heeley, Deputy Counsel on Indian Affairs for the U.S. House of Representatives Committee on Interior and Insular Affairs. The focal point of the discussion was House of Representatives bill 3724 and Senate bill 2481, the Indian Health Improvement Act of 1992. This bill identifies 59 health objectives for Native Americans and urban Indians to be achieved by the year 2000. Several objectives relate specifically to psychiatric services. Prominent features of the legislative package include the following:

1. Funding of alcohol and substance abuse programs with special emphasis on programs to prevent fetal alcohol effects and syndrome.
2. Provisions to establish programs to assure an adequate supply of Indian health professionals.
3. Special grants to Indian tribes to provide intermediate mental health services to Indian children and adolescents.
4. Establishment of an epidemiology center in each service area to monitor attainment of health status objectives and evaluate existing delivery systems.
5. Awarding of grants to Indian tribes to develop comprehensive school health education programs.

The committee believes the resources of APA and its membership can serve as a valuable resource to the U.S. Indian Health Service and Indian tribes as they strive to implement such objectives. After passage of this legislation, segments pertaining to psychiatric services will be identified and directed to appropriate APA components for study.

The *Committee of Asian-American Psychiatrists*, Jambur V. Ananth, M.D., chairperson, sponsored the Asian Forum at the 1992 annual meeting. APA President Dr. Lawrence Hartmann presented the first Asian/Asian-American Awards to Drs. Tsung-yi Lin and Lindbergh S. Sata. The forum was well attended and included the presence of Dr. Kun-Po Soo, who endowed the award, together with many members of his family.

The committee selected Joe Yamamoto, M.D., for the 1993 Asian/Asian-American Award, to be presented at a noon forum in May. Dr. Yamamoto has been a pioneer in Asian-American mental health and

treatment and "has significantly contributed toward understanding the impact and import of Asian cultural heritage in areas which have relevance to psychiatry," as specified in the criteria for this award.

The final draft of the proceedings of the International Symposium on Psychiatric Research in Asia was submitted to the APA Editorial Review Panel for its review. If approved for publication, the proceedings will be published by APA.

Dr. Francis Lu, with help from Drs. Keh-Ming Lin and Albert Gaw, finished the Asian curriculum proposal and provided it to the Assembly Committee of Minority/Underrepresented Groups for its review.

Recruitment of medical students, particularly minorities, into psychiatry is one of the priorities of this committee. Last year this committee hosted a lunch for Asian-American medical students and residents at the annual meeting, to facilitate recruitment. The committee plans to host a similar gathering in May 1993.

The committee reviewed the document *Civil Rights Issues Facing Asian Americans in the 1990s, A Report of the United States Commission on Civil Rights, February 1992*, and received council approval to request APA support (in the form of letters to the Assistant Secretary for Health, the Civil Rights Commission, and the AMA) of the following particular provisos.

Recommendation 32

Public health and other social service programs should strive to meet the specific needs (e.g., interpretation, cultural sensitivity) of low-income and immigrant Asian American communities. Federal funding for such programs should be increased.

Recommendation 33

The Department of Health and Human Services should raise the priority given to increasing the number of trained health care professionals who have the linguistic and cultural skills to serve immigrant Asian American communities. Asian Americans who meet these qualifications should be included in programs targeted at increasing numbers of minority health care professionals.

Recommendation 35

Public health data should be collected and reported separately for Asian American subgroups.

Recommendation 38

Federal, State, and local funding agencies should fund social services programs that meet the specific needs (e.g., interpretation, cultural sensitivity) of battered Asian American wives. In particular, such agencies should adopt flexible funding formulas to allow social service agencies to serve higher cost clients, such as Asian American battered wives.

The **Committee of Black Psychiatrists**, Robert T.M. Phillips, M.D., chairperson, prepared a symposium for the 1993 annual meeting, to focus on minority women in prison.

The committee tabled its plan to draft a position statement on racial harassment after learning that the council is working on a position statement on bias-related incidents.

In an attempt to become more involved in APA's efforts to promote psychiatry among medical students and to recruit residents, the committee plans to continue its liaison with the Council on Medical Education and Career Development and its components.

Dr. Diane Felder, noting the need for a component on minority children, will send a letter outlining the need for and charge to such a component, and Dr. Phillips will send the letter to appropriate components.

At the September meeting Dr. Phillips asked the members what direction the committee should take. All agreed that the committee should recruit black medical students into psychiatry, work to in-

crease the number of black psychiatrists in academia and in research, and establish a network of black psychiatrists in APA and other organizations.

The committee proposed a workshop, "Asserting African-American Values in White Organizations," for the 1993 annual meeting. Also in May 1993, in response to a request from Dr. Carolyn Haynie of the Committee on Juvenile Justice Issues, the Committee of Black Psychiatrists will work with the former on a presentation on black adolescent violence. Dr. Felder agreed to speak on the psychosocial aspects of teenage violence.

Citing the numerous problems faced by poor minority youth, particularly in mental health care, the committee agreed to broach to the Council on Children, Adolescents, and Their Families the idea of forming an APA component that will address this issue. The committee will approach other APA minority components as well.

Dr. Francis Lu asked the committee to join the Committee of Asian-American Psychiatrists' drive to recruit minorities into psychiatry and APA. As part of this effort, Dr. Lu suggested encouraging dialogue between the Council on National Affairs and the Council on Medical Education and Career Development and between the directors of the Offices of Education and Minority/National Affairs. The Committee of Black Psychiatrists will discuss this further with the Committee of Asian-American Psychiatrists.

The committee proposed noted child advocate Marian Wright Edelman for the 1993 Solomon Carter Fuller Award.

The **Committee on Gay, Lesbian, and Bisexual Issues**, Richard A. Isay, M.D., chairperson, discussed the responses by Drs. John C. Nemiah and Daniel X. Freedman to the committee's request that the word "gay" be used more frequently in both journals. The committee feels that "homosexual" is too clinical, whereas using "gay" is more consonant with APA's efforts to destigmatize and demedicalize homosexuality. Dr. Nemiah responded that "homosexual" is more scientific and will continue to be used in scientific communications. However, if authors use "gay" in editorials, commentaries, or other opinion pieces the *Journal* does not change it. The committee also felt that a letter to the search committee for the *Journal's* new Editor, requesting more minority representation on the editorial board on issues regarding editorial policy, would be desirable and appropriate.

The committee noted the intention of the Committee on Abuse and Misuse of Psychiatry in the U.S. to include a discussion of "reparative therapy" in the appendix to its draft paper on abuse. Dr. Isay has been in touch with Dr. William Womack, a member of that committee, about such a discussion, but the precise format and content are still in flux. Also regarding reparative therapy, the committee met with the Committee on Religion and Psychiatry to begin discussion about issuing a joint statement. Work on this is in progress. Dr. Robert Cabaj brought to the committee a statement on reparative therapy, with three issues that might be most effectively addressed separately: 1) APA labeling reparative (conversion) therapy as unethical, 2) a continuing effort to have reparative therapy labeled an abuse or misuse of psychiatry, and 3) finding a way to isolate the National Association for Psychoanalytic Research and Therapy of Homosexuality (NARTH), a group whose members feel conflicted homosexuals *can and should* be changed to heterosexuals. A rough draft position statement was presented to the council for information only at this time.

In preparation for APA's participation in the World Psychiatric Association Congress, the committee proposed the following position statement and resolution. Note that both were approved by the council and, subsequently, by the Joint Reference Committee and the Board of Trustees (in December 1992):

Whereas homosexuality per se implies no impairment in judgment, stability, reliability, or general social or vocational capabilities, the American Psychiatric Association calls on all international health organizations, psychiatric organizations, and individual psychiatrists in other countries to urge the repeal in their own country of legislation that penalizes homosexual acts by consenting adults in private. And further, the APA calls on these organizations and individuals to do all that is possible to decrease the stigma related to homosexuality wherever and whenever it may occur.

In conjunction with this statement, the committee also proposed the following resolution:

The American Psychiatric Association calls on the World Psychiatric Association to send the following resolution for vote at the General Assembly meeting in Brazil in June 1993: "Whereas homosexuality per se implies no impairment in judgment, stability, reliability, or general social or vocational capabilities, the World Psychiatric Association calls on its member organizations and individual members to urge the repeal of legislation that penalizes homosexual acts by consenting adults in private. And further, the World Psychiatric Association calls on these organizations and individuals to do all that is possible to decrease the stigma related to homosexuality wherever and whenever it may occur."

The *Committee of Hispanic Psychiatrists*, Fernando J. Milanes, M.D., chairperson, has written a proposal for the development of a data file on Hispanic service agencies dealing with mental health, substance abuse, and social services, as well as a compendium of individual practitioners noting their interests and areas of expertise. APA signed a contract with the National Institute of Mental Health (NIMH) to begin phase 1 of this project, which is to develop the means for collecting pertinent data.

An informal advisory committee chaired by Dr. Milanes and with Drs. Enrique Garza-Trevino, Luz Guervara-Ramos, Rodrigo Munoz, and Pedro Ruiz as members had two conference calls and one meeting. The advisory group has approved a one-page questionnaire to be sent to APA Hispanic members; it will review a questionnaire to be sent to agencies. In September the committee met with Mr. Lance Binkley, research assistant for the Hispanic Data Base Project, and Ms. Linda Roll, staff project director, to discuss the questionnaire to be sent to organizations that provide mental health care, substance abuse, and psychiatric social services to Hispanics. The committee also drafted the cover letter to be sent to these organizations. In addition, the committee drafted the proposal for phase 2 of the project and asked Ms. Roll to prepare the budget and to discuss with Dr. Juan Ramos, the project officer, the possibility of obtaining the necessary equipment to carry out the next phase of this work.

One of the committee's tasks is to propose the Simon Bolivar Award recipients. For 1993 the committee chose A. Anthony Arce, M.D.

The committee submitted a proposal for the workshop "Underinsurance and Access to Care: A Case for Hispanics" for the 1993 annual meeting. The committee discussed participation in the APA sesquicentennial and decided on a monograph titled *Hispanic Contributions Through APA's History*. Dr. Victor Rosado will coordinate the writing and production of the monograph.

Dr. Raymond Freebury and two members of the Committee on Abuse and Misuse of Psychiatry and Psychiatrists met with the Committee of Hispanic Psychiatrists. They informed the latter that members of the Mexican Foundation for the Rehabilitation of the Mentally Ill apprised their committee of the abysmal situation facing the mentally ill in Mexico. The Committee of Hispanic Psychiatrists concurred with the need to verify the allegations and, if they are found to be true, to make every effort possible to improve mental health care delivery in Mexico. The committee also agreed to recommend that the Council on International Affairs consider a site visit to these facilities in Mexico, as well as contact government officials to verify the situation and to take whatever steps are helpful to ensure that the mentally ill in Mexico are treated as humanely as possible.

The *Committee on International Medical Graduates*, Richard Balon, M.D., chairperson, recommended establishing the George Tarjan Award, given, annually if possible, to an individual who has made a significant contribution to the enhancement of the integration of international medical graduates into American psychiatry. The committee will undertake raising a \$50,000 endowment from the international medical graduate community and other sources, as approved by the Medical Director's office.

The committee feels that there are not enough international medical graduates among the senior examiners of the American Board of Psychiatry and Neurology (ABPN) and that there are discrepancies in the ABPN's recognition of psychiatric education outside the United States. The committee will explore these issues with the Task Force

to Facilitate Communication Between the APA and ABPN before submitting action items.

The committee also discussed what it considers the discriminatory practice of listing the number of international medical graduates in the programs included in the *Directory of Psychiatry Residency Training Programs* published by APA. It postponed action until after discussion with appropriate APA staff.

In a discussion about the *International Psychiatrist Newsletter*—its future, mission, and contributions by the editorial board and readers—suggestions included a residents' column, organization of articles by the editorial board, and surveys to determine more accurately the professional activities and perspectives of international medical graduates.

Ms. Gloria Chi-Fishman, director of the International Center for Accent Improvement, spoke to the committee about the advantages of modifying accents and the method used by the center to bring this about. Corporations and international organizations have sent employees to the center and paid the fee. The committee had considered a workshop on accent reduction at the 1993 annual meeting but decided on another topic, "Recent Developments in the Diagnosis and Treatment of Borderline Personality Disorder."

The committee discussed the feasibility of APA members reconstituting an international medical graduate caucus, with possible efforts again to gain Assembly representation ultimately. At present, Drs. Arthur Kranz and Nalini Juthani are international medical graduate observers to the Assembly, able to attend Assembly meetings at their own expense, but without vote and with limited voice. After meeting with representatives from the Committees of Asian-American Psychiatrists and Hispanic Psychiatrists, the committee decided to support the formation of an international medical graduate caucus.

The committee agreed to formulate an "IMG Manifesto," a white paper on how APA can assist international medical graduates.

In response to Dr. Silvia Olarte's question on how the council might help the committee to improve its work, the committee suggested a joint meeting with committees whose work overlaps with that of this committee, e.g., with the Committee of Asian-American Psychiatrists and the Committee of Hispanic Psychiatrists, during the annual and fall meetings. The committee also felt that the council should help recruit young members to this and other components and wants to receive the council's minutes regularly.

The committee felt strongly that APA members who are international medical graduates be contacted early and their representation sought whenever APA sends groups to establish relationships with other countries' psychiatric societies. The Committee on International Medical Graduates asks that APA international medical graduate members be informed of proposed APA delegations to other countries because the committee feels that international medical graduates have expertise potentially helpful in establishing contacts and aiding effective liaison. The committee will prepare a list of international medical graduates with particular expertise and will make that list available to the Council on International Affairs and other pertinent APA components. Note that the council suggested that its chairperson discuss this informally with the chairperson of the Council on International Affairs, rather than presenting it as an action item to the Joint Reference Committee. This has occurred.

The *Committee on Psychological Aspects of Nuclear Issues*, Stephen Shanfield, M.D., chairperson, proposed a joint symposium, cosponsored by the Council on Children, Adolescents, and Their Families, the Committee of American Indian, Alaska Native, and Native Hawaiian Psychiatrists, and the Corresponding Task Force on Psychiatric Dimensions of Disasters, on psychological responses to nuclear contamination. Transport and storage of nuclear waste is just one of the subjects for which both technical and psychological issues are obvious. The committee learned, at the time this report was being prepared, that the Scientific Program Committee did not accept the proposed symposium. An agenda item for the next committee meeting, and for the Council of National Affairs at its winter 1993 meeting, will have to do with how to help extend the membership's, and the Scientific Program Committee's, appreciation of the breadth of psychiatric issues in this committee's charge and the importance of such issues for our profession, as well as our entire society.

The committee also has begun to consider a workshop or symposium

sium for the 1994 annual meeting, about both the positive and negative aspects of the military-industrial-academic-governmental complex that has served as the main proponent for both military and peaceful nuclear development.

Dr. Shanfield and Dr. Jerrold Post are making progress on a model curriculum on political psychology for psychiatry residents. The authors are developing a module that could be made available to interested residency training programs.

The committee was invited to visit Minsk, Belarus, to study the psychosocial impact of the release of radioactive material as a result of the destruction of a reactor at the Chernobyl nuclear power plant. Dr. Vamik Volkan has been in touch with Spartan Polsky, Director of the Laboratory of Ethnic Geography at the Minsk Pedagogical Institute. The committee plans to accept this invitation for a site visit to Belarus and will write a paper on its findings, analyses, conclusions, and recommendations. The committee also plans to prepare a symposium on those observations and conclusions that will be submitted for presentation at an APA annual meeting.

The Committee on Psychological Aspects of Nuclear Issues also discussed topics for a 1994 symposium, tentatively deciding on the topic of ethnic unrest. Such a symposium is quite consonant with the overall task of the committee, which is to educate the membership on the broad range of nuclear issues. In this post-Cold War period, there are 35 *intrastate* wars and *no* interstate wars. These wars are largely due to ethnic conflict that has the potential to escalate to larger conflagrations, which can include the use of nuclear weapons.

The committee had three guests at its September meeting: James Riccio of Public Citizen, Nuclear Mass Project, who discussed public policy and low-level nuclear waste; Steve Doley of the Nuclear Control Institute, who discussed nuclear proliferation; and William Arkin, Ph.D., of Greenpeace, who discussed the proliferation of nuclear weapons in a changing world.

The *Committee on Religion and Psychiatry*, Richard Thurrell, M.D., chairperson, recommended Paul R. Fleischman, M.D., an APA member and author of *The Healing Spirit* and other writings on the interface of psychiatry and spirituality, for the 1993 Oskar Pfister Award. Financial support for the award seems likely through an interest-earning fund to be set up within APA that would accept earmarked contributions from various sources, including a specific bequest from the Harding Family Fund. George T. Harding IV, M.D., a corresponding member of this committee, was pivotal in arranging this financial support. Half of the honorarium will continue to come from the Association of Mental Health Clergy; APA's fund will pay the other half and administrative costs.

In November 1991 the Assembly referred an action paper on Christian psychiatry to this committee. The paper's author, Dr. Allen Kayser, met with the committee, which decided to consult the Ethics Committee and other relevant components before sending its comments to the Assembly.

The committee's embryonic book project, informally titled *What Every Psychiatrist Should Know About Patients' Religious Beliefs*, is taking shape. Draft summaries of Catholic, Bahai, Unitarian, Southern Baptist, Santeria (a South American Catholic-native faith), and Zen Buddhist viewpoints have been submitted for committee review. Still to come are summaries of Episcopalian, Methodist, Sufi, Moslem, Alaskan native, Seventh-Day Adventist, Jewish, Mormon, and Hindu viewpoints.

As instructed by the committee, Dr. Thurrell had written to the Task Force on DSM-IV, Dr. Allen Frances, chairperson, supporting the general idea that psychospiritual disorders be included in the formerly designated "V codes" of DSM-IV, in the manner suggested by Dr. Francis Lu in September 1992.

The "Guidelines on Possible Conflict Between Psychiatrists' Religious or Ideologic Commitment and Psychiatric Practice," which the committee wrote, has been helpful in responding to inquiries and complaints from APA members and other concerned citizens.

The committee discussed the APA "Clergy Kit" with Mr. Walter Hill of APA's Division of Public Affairs. There will be a revised edition for the 1992 Mental Illness Awareness Week.

A primary ballot of six candidates for the 1994 Oskar Pfister Award was drafted, and material about each candidate will be circulated by committee members for a final recommendation in May 1993. The committee discussed the practice of alternating an awardee

who is an APA member and one who is not, but it did not reach a specific policy decision. As in previous years, a larger list of potential awardees was "rolled ahead" for 1995 and beyond.

The Committee on Gay, Lesbian, and Bisexual Issues and this committee discussed "reparative therapy," which some religious leaders and denominations advocate for supposed problems of gender identity. There will be further discussion for a possible APA position paper on the subject.

The committee was reminded that local and district branch activities in the area of religion and psychiatry, especially relations with local clergy, have been less prominent among committee activities, and the committee agreed that the promotion of local activities and interest should not be neglected. Similarly, the committee will pay attention to educating residents on the importance of patients' religious backgrounds.

Reverend Clark Aist, Ph.D., outlined the nature of the Association of Mental Health Clergy and discussed some of its activities. He also discussed the history of the Committee on Religion and Psychiatry and the Oskar Pfister Award.

The *Committee on Women*, Gail E. Robinson, M.D., chairperson, urged that APA join the Planned Parenthood Federation of America to urge Congress to pass HR-3090, which restores the family planning program to the standard that prevailed until the "gag rule" was imposed. Dr. Sabshin indicated that APA would continue to advocate for HR-3090 or its equivalent.

The committee continues to urge that more attention be focused on the problems in recruitment and promotion of women in academic psychiatry, as well as the need to specifically include *women's* mental health issues in research endeavors. The committee also may arrange to meet with the women's Congressional caucus in order to emphasize the lamentable lack of attention to women's mental health.

Sexual harassment in the workplace continues to make the news. Dr. Margaret Jensvold gave the committee an update on her lawsuit against NIMH. According to her, there are many lawsuits and Equal Employment Opportunity Commission complaints against NIMH. There is concern that NIMH continues to misrepresent the statistics on the number and status of women it employs and that retaliation is increasing against women who complain.

The committee was asked to respond to the American Association of Plastic Surgeons' request for support on the issue of breast implants. That association is worried that the U.S. Food and Drug Administration (FDA) has banned implants for political reasons. The association believes that at present there is no clear relationship between implants and negative results. In the course of the committee's discussion, members voiced the beliefs that these devices were marketed without adequate information about procedural safety, that there should be no interference with women's right to choose implants provided they have adequate information about safety, and that the FDA has not properly established the safety of this procedure or the lack of it. The committee feels that the FDA should study women who have implants to check for safety. If there is evidence of their safety, the FDA should conduct further testing by studying women who request breast reconstruction after surgery and then those who request breast augmentation. The committee's opinion is that such a study should include appropriate psychological and psychiatric assessment to document the effects of breast implants on the recipient's sense of well-being.

There were further requests from the American Association of Plastic Surgeons to try to convince this committee to support women's right to choose breast implants. The committee decided to stay with its original statement that women should do this only if they are able to give a bona fide informed consent. The nature of the research in breast implants appears not to be at a stage where a comprehensive informed consent is possible.

Last year the committee proposed a position statement on the advancement of women and underrepresented minorities. Submission of the proposal was postponed because the Committee on Research Training was preparing a paper on the same subject. In the meantime, Dr. Nada Stotland suggested revisions to the position statement that the committee incorporated in another draft. The Assembly Committee of Minority/Underrepresented Groups raised other concerns that prompted the Committee on Women to postpone further action for now. Instead, the committee will obtain information on statistics on

advancement and recruitment in federal agencies from Mr. Jay Cutler, Director of the APA Division of Government Relations, will contact chairpersons of departments of psychiatry to ask if they need help in finding eligible women candidates, and will contact groups in the AMA with similar concerns.

The committee again is planning for an activity center at the 1993 annual meeting. As before, there will be meetings at the center. Topics for 1993 include "Passing the Board Examinations," "Women in Academia" (including becoming active in research and in publishing), and "Reasons for Being Active in Organized Medicine."

Senior Deputy Medical Director Dr. Carolyn Robinowitz suggested that this committee might be interested in undertaking a research project on therapists who have treated patients who were previously sexually abused by someone who was in a fiduciary relationship with them. She suggested that the benefits of the survey could include the future development of educational material for such therapists. APA would be able to provide staff support. It was suggested that the Psychiatrists' Purchasing Group might be interested in funding such a project. Trying to proceed with funding about which there could be no claim of a potential conflict of interest in the research was also discussed.

The committee discussed areas that might be included in such a survey. These included prevalence; demographic characteristics of the patient, fiduciary agent, and other therapists who were contacted or who refused to provide treatment; numerous questions about the abuse per se (e.g., time between the abuse and the presentation at the second therapist, amount of time in nonabusive therapy before the earlier abuse was revealed); questions about the second therapist's attitude toward dealing with such cases (e.g., hesitation about treatment, willingness to treat if litigation was still in process or if there were criminal statutes or reporting requirements in that state); and questions about the psychological impact on the patient and countertransference feelings in the second therapist.

Ms. Phyllis Greenberger of APA's Division of Government Relations provided the committee an update on the status of various issues, such as freedom of choice, family leave, and RU-486.

A number of proposals concerning the committee's participation in the sesquicentennial celebrations were made. In general, these proposals encompass both looking at the history of women in psychiatry and looking at ongoing developments and issues for women. Several concrete suggestions included establishing a traveling exhibit that would include the history of women in psychiatry and that could go to various medical schools for use as a tool for recruitment of female medical students; providing various APA teaching tools, such as videotapes on domestic violence or date rape, which could be sent to the district branches with suggestions that they offer symposia for such groups as churches, parent-teacher associations, and schools inasmuch as they would illustrate how psychiatry is relevant to basic community needs; inviting Ms. Anita Hill to speak at the conference to highlight the committee's, the profession's, and society's continuing concern about sexual harassment; and organizing a symposium or workshop on the history of women in psychiatry.

The committee will work with the Committee on Gay, Lesbian, and Bisexual Issues and the Committee of Black Psychiatrists over the next 9 months to develop an action item concerning societal violence. The committee will also work with the Committee on Family Violence to draft an action item on domestic violence.

Dr. Frederick Goodwin, Director of NIMH, met with the committee and told members about a number of initiatives underway concerning the amalgamation of NIMH and the National Institutes of Health. These include, among others, a women's health consortium that will take an interest in women's mental health issues and women's careers, the presence of a new professor of psychiatry, Dr. Freda Lewis-

Hall, who will focus on minority and women's issues, and a new role for Dr. Ellen Leibenluft. There will be a major focus on getting more women involved in research by extending the tenure process beyond the current 7 years. There will also be increased interest in services research, which may be of interest to more women.

The *Task Force on Psychiatric Dimensions of Disasters*, Robert J. Ursano, M.D., chairperson, was established following the disbanding of the Corresponding Task Force on Psychiatric Dimensions of Disasters, which had been chaired by Robert Pynoos, M.D., who continues as a member of the new task force. The importance of psychiatric responses to disasters was again highlighted by Hurricane Andrew. The role of APA in response to this disaster was reviewed, as was material sent out to district branches in Florida. It is important to note that disasters involve community, public, and federal (military and Department of Veterans Affairs) psychiatric resources. These groups often represent the underserved areas of the population, which can be hardest hit by a disaster and have limited resources with which to respond. Disasters extend over time and are best thought of as the result of both preimpact and postimpact events. An earthquake in the Sahara Desert is not the same as an earthquake in San Francisco. A disaster affects, and is affected by, both the communities and the individuals involved.

The task force reviewed its goals and emphasized education, resource development, and coordination among district branches, APA components, and extra-APA organizations as its primary modes of action. And the task force will be reviewing APA's own disaster plan! It would be helpful for APA's Office of Public Affairs to be involved in the rapid distribution of material and to facilitate the availability of resource psychiatrists to disaster areas, both in direct clinical work and also in public education and reassurance, i.e., through the media. The task force sees one of its roles as assisting APA with resources for training psychiatrists and for psychiatrists to use in training other health care workers. We need to obtain from the Pan American Health Organization an available series of slides and teaching material titled "Mental Health Management in Disaster." These should be distributed to all district branches. Moreover, an article on disasters, "Psychosocial Consequences of Disasters," published in 1992 by the World Health Organization, should be available from the Division of Public Affairs and distributed to all district branches.

As for educating the public, the task force will write the text for a "Let's Talk Facts" brochure on disaster. And the task force also proposed expanding APA's consultation service to include consultation in times of disaster and consultation in the design of the mental health components of disaster plans. The consultation service would be self-financed and would be designed to provide a resource to work with district branch representatives to facilitate the development of local skills and to expedite coordination and access. The task force plans to write a guideline for consultation that will serve as a discussion topic for the task force workshop.

Another suggestion is that APA add a section on disasters to its "Awareness Guides" for educators, clergy, the media, and health care professionals. These guides should be available in English and Spanish.

The task force submitted a proposal for the course "Basic Traumatic Stress Care," which will train practitioners in the basic elements of care for patients diagnosed with various types of traumas. If this course is successful, a follow-up course, "Advanced Traumatic Stress Care," which will focus on consultation to organizations, will be offered.

Because disasters cut across many areas in APA and in the United States, APA might wish to consider establishing a presidential commission to deal with the topic of disasters in a positive, proactive, and visible manner.

The Council on Psychiatric Services

James T. Barter, M.D., Chairperson

The Council on Psychiatric Services is committed to furthering the delivery of high-quality psychiatric services. Psychiatrists, whether working in the private sector or the public sector, must constantly wrestle with an overburden of rules and regulations from governmental agencies, third-party payers, and managed care organizations. It is difficult to continue to promote easy access to quality care and services in such circumstances. Looking for alliances in this difficult climate, the council wants to establish ongoing communication with the new federal Center for Mental Health Services, under the auspices of the Substance Abuse and Mental Health Services Administration (SAMHSA). To that end, it began a dialogue with the center's Acting Director, Frank Sullivan, Ph.D., at its September meeting and organized additional meetings between Dr. Sullivan and psychiatric leaders in the public sector during the Institute on Hospital and Community Psychiatry. On the private sector front, after much discussion, the council agreed with the Committee on Private Practice that, in this time of economic and regulatory change, a transfer of that committee to the Council on Economic Affairs was in order. This was accomplished; it is hoped that placement of the committee within the Council on Economic Affairs will facilitate communication with other economic components, such as those addressing managed care, codes and reimbursement, and financing and marketing. With the belief that issues involving treatment and practice in a private practice setting remain a major priority, the transfer was made on the condition that the question of placement of the Committee on Private Practice within the APA structure would be periodically revisited.

The following reports of the council and its component committees reflect psychiatry's commitment to our patients, our society, and ourselves.

The *Consortium of Chairpersons of APA Components on Public Psychiatry*, J. Frank James, M.D., chairperson, was established by the Board of Trustees in December 1990 and charged with identifying deficiencies in the delivery of care to groups with special needs. It was also charged with documenting existing or hypothetical models that address such deficiencies in a cost-effective and clinically sound manner. Accordingly, the group prepares information for the APA leadership that may be used for advocacy and other purposes to benefit the psychiatric care delivery system. The consortium reports to and is accountable to the Council on Psychiatric Services.

The consortium includes the chairpersons of the following components: Committee on State and Community Psychiatry Systems, Task Force on the Homeless Mentally Ill, Committee on Chronic Mental Illness, Committee on Psychiatric Disability and Rehabilitation, Committee on Veterans' Affairs, Committee on Psychiatric Services in Jails and Prisons, Committee on Psychiatric Services in the Military, Committee on Psychiatric Services for Mentally Retarded and Developmentally Disabled Persons, Committee on the Practice of Psychotherapy, State-University Collaboration Project, Corresponding Task Force on Geriatric Psychiatry in the Public Mental Health Sector, Committee on Chronically Ill and Emotionally Handicapped Children, Assembly Committee on Community Psychiatry, and Assembly Committee on Public Psychiatry.

The consortium has stressed the need for access to treatment and care for the severely mentally ill and the need to ensure such access in any national health care plan that may emerge. The consortium has also called attention to the need to involve more psychiatrists in the public sector and, hence, the need for incentives for psychiatrists to seek positions or volunteer services in the public sector, as well as recognition of their efforts. Over the past year the consortium has generated many recommendations, which are now in various states of study, approval, and implementation. Most important, while the consortium called for support for universal coverage for all mental illness on a parity with coverage for physical illness, it particularly promoted efforts to ensure that severe and persistent mental illness

would not be discriminated against by exclusion or biased limitations. The consortium believes that such coverage should be integrated with appropriate systems of peer-governed utilization review. Aware of efforts by national groups, including the new Center for Mental Health Services, to define the concepts of severe and persistent mental illness, the council and consortium believe that APA should develop its own definition. Additionally, the council and consortium asked the Committee on Chronic Mental Illness to develop guidelines for access to and continuity of treatment for severe and persistent mental illnesses.

The *Committee on the Practice of Psychotherapy*, William Hurt Sledge, M.D., chairperson, reviewed drafts of practice guidelines for eating disorders and major depression and availed itself of the opportunity to be continually involved in the practice guideline review process. The committee explored additional ways to increase psychotherapy training in psychiatric residency training programs. As one way to reach the practicing psychiatrist, the committee sponsors clinical case conferences at the APA annual meetings. In addition, the committee is working with the Scientific Program Committee to influence the organizers of industry-sponsored symposia to include psychodynamic psychotherapy as one of the treatment options for the topics presented (anxiety disorders, anxiety and the medically ill, etc.). The committee will also explore the possibility of presentations on managed care and individual and group psychotherapies at future annual meetings.

The *Committee on Chronic Mental Illness*, Stephen Goldfinger, M.D., chairperson, made known its support for a strong mental health focus within the new SAMHSA, which emerged when the National Institute of Mental Health, National Institute on Drug Abuse, and National Institute on Alcohol Abuse and Alcoholism were moved under the auspices of the National Institutes of Health. The committee has continued to analyze data from the most recent APA biographical survey to capture information concerning the treatment of persons with chronic mental illness.

The Committee on Chronic Mental Illness has maintained its interest in HIV/AIDS as it affects those with chronic mental illness. Stimulated by the committee, and with the council's encouragement, the Consortium of Chairpersons of APA Components on Public Psychiatry will arrange a meeting with representatives of the APA Commission on AIDS and the National Association of State Mental Health Program Directors to develop strategies for the most effective methods for fighting the effects of this disease on psychiatric patients. In September the committee spent considerable time debating definitions for the population with severe and persistent mental illness and the possibility of developing guidelines for treatment of this population.

The *Committee on Psychiatric Disability and Rehabilitation*, Kenneth Terkelsen, M.D., chairperson, addresses matters of national law and regulations affecting children and adults with disabilities, including physical disabilities complicated by psychiatric conditions. During 1992 it provided commentary relating to the Social Security Administration's modernization project, regulations on evaluation of HIV infection for disability, and proposed rules on vocational rehabilitation. Further, it focused on the implementation of the Americans With Disabilities Act and monitored the development of practice guidelines and of revisions to American Medical Association guidelines for evaluation of permanent disability by physicians.

Together with the APA Office of Psychiatric Services, it cosponsored with the Social Security Administration an annual meeting workshop titled "Disability Evaluation Under Social Security: A Presentation for Treating Psychiatrists."

The *Committee on Psychiatric Services for Mentally Retarded and Developmentally Disabled Persons*, Ludwik S. Szymanski, M.D., chairperson, continued to build on the work of the previous Task

Force on Psychiatric Services to Mentally Retarded Adults. Additionally, the committee's work included development of a draft curriculum and networking with a variety of organizations concerned with standards, emerging legislation and regulations, and education. It also maintained contact with the state departments of mental health and/or mental retardation. Through a brief article in *Psychiatric News*, the committee has begun a roster of psychiatrists interested in this subspecialty area and has identified a number of experts who may serve as faculty.

The *Committee on Psychiatric Services in Jails and Prisons*, Henry C. Weinstein, M.D., chairperson, considered ways to increase attention to psychiatric services provided in these settings. The committee encouraged district branches to include representation of the corrections psychiatry field in their public psychiatry components. The committee further explored the feasibility of a resource pool of individual experts and of materials that can be made available to psychiatrists, other interested professionals, and the public. The council commended the support of the task force, and especially that of Dr. Weinstein, for a report of the National Alliance for the Mentally Ill and Public Citizen's Health Research Group, *Criminalizing the Seriously Mentally Ill: The Abuse of Jails as Mental Hospitals*, which became available in September 1992.

The *Committee on State and Community Psychiatry Systems*, Steven E. Katz, M.D., chairperson, continued to encourage APA district branches and Areas to involve themselves in their states' efforts to develop annual comprehensive plans for services to the serious and persistent mentally ill in accordance with Public Law 99-660. Public Law 102-321, which supersedes Public Law 99-660, now mandates "appropriate review" with a penalty of 10% of the state's block grant if the mandate is not fulfilled. The committee will continue monitoring this legislation and will seek clarification of implementation processes.

The committee continues its efforts to have its "Guidelines for Psychiatric Practice in Community Mental Health Centers" recognized by the Joint Commission on Accreditation of Healthcare Organizations. Additionally, the committee has recommended that APA encourage state psychiatric societies and district branches to advocate adoption of these guidelines by state mental health authorities. A model job description for medical/clinical directors in state mental health authorities, developed by this committee, was approved by APA in late 1991. Now in development is a job description for medical directors in state psychiatric facilities.

The committee has focused attention on federal funding of services research, on the federal mandate to define serious mental illness for adults and children, and on how determinations of prevalence and incidence will be made. The committee is strengthening liaisons with the National Association of State Mental Health Program Directors and the Assembly Committee on Public Psychiatry and will collaborate with the Consortium of Chairpersons of APA Components on Public Psychiatry to present a workshop on "Guidelines for Psychiatric Practice in Public Inpatient Hospitals."

The *Committee on Psychiatric Services in the Military*, Harry C. Holloway, M.D., chairperson, called to the attention of the council the development of mental health standards by the U.S. Department of Defense's Office of Health Affairs and prompted APA to offer expertise and comment. Working with APA's Division of Government Relations, the committee provided valuable advice concerning CHAMPUS, the downsizing of the military force and military medicine, the Department of Defense pilot program for training psychologists to prescribe medications, whistleblower issues, and military and Public Health Service pay issues. The committee supports a proposal for a separate branch for military psychiatrists within APA and encouraged continued discussion by appropriate APA components.

The *Committee on Veterans' Affairs*, Frederick Guggenheim, M.D., chairperson, continued to provide advice to the Division of Government Relations concerning education, budget, and pay issues. Members continued their annual visits to members of Congress, and plans were implemented to enhance communication with veterans' service and consumer organizations. Planning was initiated for a component workshop to be presented at the 1993 annual meeting that will focus on posttraumatic stress disorder, geriatrics, substance abuse, and chronic mental illness.

Issues explored by the committee and on which it provided commentary and recommendations include the U.S. Department of Vet-

erans Affairs (VA) support for mental health and addictive disorders, extension of the APA State-University Collaboration Project to include VA neuropsychiatric hospitals and psychiatry, and the profound need for additional resources for VA neuropsychiatric hospitals. Additionally, the committee noted that the VA has not been mentioned in any of the national health care plans under consideration by Congress. It called on the APA Board of Trustees to highlight in all possible forums the significant benefits to the nation from the VA affiliations with schools of medicine and how the VA system, particularly specialty care, could fit into plans for universal access to care.

The *Committee on the H&CP Service*, Gail Barton, M.D., chairperson, met in the fall of 1992 to review membership trends and current service programs. The committee is pleased that in spite of these difficult economic times membership is holding steady with 750 member facilities. Distribution of the *Hospital and Community Psychiatry* journal through the service accounts for 35% of the journal's current circulation. The service's videotape and film library now includes nearly 200 titles. Ian Alger, M.D., Video Consultant, reviewed and selected the 36 new titles in the past year, and the 1993 videotape and film catalog is available on request. This unique library of blockbuster mental health videotapes continues to be a very popular program. The Psychiatric Placement Service had a successful year, placing psychiatrists in facilities and serving as a resource to members interested in the psychiatric employment field. The committee provides oversight to the placement service, which was established by the H&CP Service in 1986 under the guidance of H. Richard Lamb, M.D. Since that time the placement service has found new positions in a variety of settings for nearly 100 psychiatrists. The vast majority of placements have been in hospitals, followed by group practices and community mental health centers. The quarterly *H&CP Service Update* was sent for the first time in 1992 to all 8,500 mental health professionals in service facilities who were identified as "key personnel." The response to this newsletter has been encouraging, and the service plans to expand the *H&CP Service Update* to include more clinical information.

The *Institute on Hospital and Community Psychiatry Program Committee*, David A. Olenik, M.D., chairperson, continued its practice of providing current, practical interactive sessions at its meeting in Toronto, Ont., Canada, Oct. 23-27, 1992. The institute's theme, "Partnerships for Mental Health: Access, Quality, Cost," was reflected in a range of topics, such as managed care, universal access to health care, comparisons of the Canadian and U.S. systems, adaptive tasks for psychiatry, and international mental health care delivery programs. Sessions on administrative psychiatry, case management, incest, substance abuse, geriatric care, and multiple personality disorders were especially well attended. Thirteen professional and allied groups held meetings in conjunction with the institute. Alan M. Elkins, M.D., is chairperson of the program committee for the 1993 institute, which will be held in Baltimore on Oct. 8-12.

Established in 1989, the *Task Force on the Homeless Mentally Ill*, Frederic I. Kass, M.D., chairperson, published two interim reports, "General Directions for Public Policy in Behalf of the Mentally Ill Among the Homeless Population" (1990) and "Homeless Families and Children: A Psychiatric Perspective" (1991). This year the task force completed *Treating the Homeless Mentally Ill*, a book that includes recommendations and a summary of developments in psychiatric services for the homeless mentally ill in the last several years. During its tenure, the task force initiated several workshops for district branches at APA annual meetings, with the goal of facilitating exchange of information, know-how, and enthusiasm among district branches interested in developing local programs for the homeless mentally ill population.

In December the Board of Trustees expressed its appreciation to members of the task force and disbanded it. To ensure an ongoing mechanism to fulfill APA's commitment to the mentally ill persons among the homeless population or those at risk of homelessness, the Board created a standing committee within the Council on National Affairs. The new Committee on Homelessness is charged with advocacy related to the psychiatric aspects of many factors of homelessness, such as mental illness, substance abuse, HIV/AIDS, treatment and service delivery, and legal and social issues.

The *Task Force on Family/Systems Therapy*, Fred Gottlieb, M.D., chairperson, created linkages to other APA components, raising consciousness about family/systems therapy in terms of considerations

for treatment, reimbursement, and education. The task force has reviewed previously published APA documents on the topic and has developed a framework for approaching current issues that encompass definitions, considerations for treatment, education, practice (including the possibility of practice guidelines), third-party payment issues (including coding and reimbursement), and research.

The *Task Force on Clinician Safety*, William Dubin, M.D., chairperson, continued its study of the issues, including facilitation of a milieu that would minimize violence, threats, and assaults on treat-

ment providers and involvement of legal authorities in threats. The task force completed its report, which is scheduled for publication in the spring of 1993. In December the Board of Trustees expressed its appreciation to the task force and noted that it would disband in June 1993.

It has been a busy and rewarding year. The council anticipates several challenges in the coming year relating to access to services, quality of care, and the strength of psychiatry's contribution in the many areas that touch on treatment of services.

The Council on Psychiatry and Law

Paul Appelbaum, M.D., Chairperson

The Council on Psychiatry and Law has come to closure on several projects this year.

The Patient Self-Determination Act (PSDA) was passed by the United States Congress in the wake of the U.S. Supreme Court's decision in *Cruzan v. Director, Missouri Department of Health* and became effective Dec. 1, 1991. In *Cruzan* the Supreme Court implicitly endorsed the right of individuals to make decisions regarding the termination of life-sustaining treatment while also upholding states' authority to impose reasonable procedures to ensure the reliability of these decisions. The PSDA arose from Congress's interest in promoting the use of advance directives as a means of facilitating reliable patient involvement in decisions regarding the withdrawal of life-sustaining care. Passage of the PSDA has raised considerable concerns on the part of some psychiatrists and psychiatric hospitals about implementation. At the same time, many psychiatrists and facilities remain uninformed about the PSDA or unaware that the provisions of the PSDA apply not only to medical settings but also to psychiatric settings. To help APA members understand the implications of the PSDA, the council has prepared a proposed resource document, "The Patient Self-Determination Act: What Every Psychiatrist Should Know." It is intended that the document, drafted in an accessible, question-and-answer format, will be made available to all interested psychiatrists and facilities through the district branches. The document has been approved by the Joint Reference Committee, which has recommended approval to the Board of Trustees. (The document will be sent to the Assembly for information.)

Psychiatric sexual misconduct with patients is a serious problem that has rightfully become of paramount concern to the profession and to legislators and other policy makers. Organized psychiatry has provided ethical sanctions and instituted educational programs in an effort to eliminate sexual misconduct. More recently, legal initiatives have been taken to address the problem. Several states have enacted legislation, generally acting to criminalize sexual misconduct and to mandate reporting of offenses; many others are actively considering doing so. To assist district branches in states that are considering legislation, the council has developed (in conjunction with the Committee on Women) a proposed resource document on psychiatrist-patient sex. The document 1) briefly reviews the scope of the problem of sexual misconduct, 2) summarizes the profession's past efforts to address misconduct, 3) reviews the major categories of legislation that have been proposed or enacted—mandatory reporting and criminalization—and 4) briefly summarizes other areas of possible

legislative initiatives. The purpose of this document is to review legislative initiatives that district branches may consider as means of more effectively curbing sexual misconduct. The document has been approved by the Joint Reference Committee, which has forwarded it to the Assembly and the Board with a recommendation for approval.

Working with the Task Force on DSM-IV, the council is developing a legal caveat to be included in DSM-IV. This caveat will provide a warning that DSM-IV is a tool to be used in aiding clinical judgment and that these diagnoses should not be applied mechanistically by nonclinicians. It will distinguish the definition of "mental disorder" for psychiatric purposes from "mental disease or defect" for legal purposes. It is anticipated that this draft will be ready for review by the council at its May 1993 meeting.

A subgroup of the Council on Psychiatry and Law and the Council on Aging has been studying the responsibilities of psychiatrists in connection with patients' driving ability. This group has focused its concerns on the narrow issue of patients affected with dementia.

During the fall component meetings the council met jointly with the Commission on Judicial Action concerning the Americans With Disabilities Act. Chai Feldblum, J.D., one of the primary authors of the act, was a guest at this meeting. There are numerous ways in which the psychiatric community will be affected by the legislation, in particular the employment of residents and mental health insurance coverage. The council will work with the Council on Medical Education and Career Development to address some of these concerns.

The *Committee on Confidentiality*, Renee L. Binder, M.D., chairperson, is working to produce a videotape for use by psychiatrists in dealing with complicated ethics/confidentiality issues. The videotape will consist of a number of vignettes followed by discussion of the issues and ways in which they might be resolved. Once completed, the videotape will be distributed to the district branches and to residency training directors.

The *Task Force on Consent to Voluntary Hospitalization*, Francine Cournos, M.D., chairperson, has completed the final draft of its report. The report focuses on the issues raised in *Zinerman v. Burch*, which raised questions about the legitimacy of consent given by patients who may be incompetent at the time of voluntary hospitalization and suggested that those patients should be dealt with by involuntary commitment. The draft has been approved by the Joint Reference Committee, which has forwarded it to the Assembly and the Board with a recommendation for approval.

The Council on Research

Herbert Pardes, M.D., Chairperson

The Council on Research, in conjunction with its components, is the body within the Association that seeks to ensure the continued expansion of psychiatry's science base and represents the psychiatric research community for the association. It focuses on science policy, research training, and scientific assessment (integrating and disseminating scientific information to the clinical community), and it oversees research studies conducted by the Association. The council has been carefully monitoring science policy issues, particularly those concerned with animal research, research appropriations, conflict of interest issues, training and infrastructure needs, and funding of child psychiatry research. Many of these issues and those of the research components are addressed in the column "News from the Council on Research" in *Psychiatric Research Report*, the Office of Research quarterly newsletter. Individuals who would like to receive *Psychiatric Research Report* should contact the APA Office of Research.

The council was delighted to meet and continue its annual discussions with key leaders of the federal research agencies, including the former Alcohol, Drug Abuse, and Mental Health Administration (ADAMHA), the National Institute of Mental Health (NIMH), the National Institute on Alcohol Abuse and Alcoholism (NIAAA), and the National Institute on Drug Abuse (NIDA). Over the years these meetings have provided an opportunity for informal collaboration and have identified issues of mutual concern. This year's discussions focused largely on the reorganization of ADAMHA, which was effective Oct. 1, 1992. The reorganization moved the research programs of NIMH, NIDA, and NIAAA to the National Institutes of Health. Service and prevention programs are now part of the Substance Abuse and Mental Health Services Administration (SAMHSA). The council agreed that continued communication is essential as the reorganization is fully implemented. Other topics addressed with the agencies included the psychiatric research infrastructure, peer review, research training, health services research, knowledge transfer, and research appropriations.

The council collaborated with the APA Division of Government Relations and the Joint Commission on Government Relations on a number of important issues. The council was particularly concerned with ADAMHA appropriations and the substantial funding cuts that have affected science generally. In addition, the council voiced concern about the public image of psychiatry, conflict of interest issues, and forthcoming regulations and reports defining severe/serious mental illness for adults and children.

The council administers two APA awards—the Lilly Psychiatric Research Fellowship and the Kempf Fund Award for Research Development in Psychobiological Psychiatry. The council appointed Ronald Rieder, M.D., Gary Tucker, M.D., William Lawson, M.D., Ph.D., Joshua Simon, M.D., and Charles Nemeroff, M.D., to review the 1993 applications and select the winners. The winners of the 1992 Kempf award were Stephen R. Zukin, M.D. (senior awardee) and Daniel C. Javitt, M.D. (junior awardee). Shitij Kapur, M.D., received the Lilly Psychiatric Research Fellowship.

Following are details of the activities of components under the Council on Research.

The *Committee on Research on Psychiatric Treatments*, John Kane, M.D., chairperson, continues to assess and respond to scientific issues regarding psychiatric treatment and is participating in the extensive review of APA practice guidelines. The committee recently published "Psychopharmacological Screening Criteria" in the *Journal of Clinical Psychiatry* (1).

The *Task Force on Electroconvulsive Therapy*, Richard Weiner, M.D., chairperson, monitors proposed regulations published by the U.S. Food and Drug Administration (FDA) reclassifying ECT devices from class III (requiring premarket approval) to class II (requiring a performance standard) for use in treating severe depression. The task force continues to encourage the FDA to modify its regulations to

include mania and schizophrenia in this reclassification and to clarify the meaning of "severe depression."

The *Task Force on Nicotine Dependence*, John R. Hughes, M.D., chairperson, was established by the Board of Trustees on a joint recommendation by the Committee on Research on Psychiatric Treatments and the Council on Addiction Psychiatry. The task force is charged with educating psychiatrists regarding the seriousness of nicotine addiction and the need for treatment, assessing current scientific information in the field, identifying reimbursement issues, developing recommendations for training, and establishing a general research agenda. The task force plans to commence these efforts by drafting a position statement and an editorial to be submitted to the *American Journal of Psychiatry*. Other new projects under consideration are a survey of residency programs to determine training in nicotine addiction and a workshop at the annual meeting of the American Association of Directors of Psychiatric Residency Training.

This issue of the *Journal* highlights the recent work of the *Steering Committee on Practice Guidelines*, John McIntyre, M.D., chairperson, by presenting APA's first practice guideline and an editorial on the goals of the practice guidelines effort. The practice guideline on major depressive disorders will be the next to appear in the *Journal* and will be followed by additional guidelines currently in development (on inpatient and outpatient evaluation, bipolar disorder, geriatric care, schizophrenia, and substance abuse). Future guideline topics under consideration are Alzheimer's disease and major depression in children and adolescents; the latter will be jointly produced with the American Academy of Child and Adolescent Psychiatry. Various dissemination strategies for APA guidelines are under consideration, including participation in a project by the American Medical Association to produce a CD-ROM (compact disk, read-only memory) of all existing treatment guidelines.

In September 1992 a methods conference titled "Challenges in Producing Psychiatric Practice Guidelines" was held, with partial financial support from NIMH. The chief problem addressed was how to produce guidelines on topics for which the data base is incomplete, such as borderline personality disorder. The development of a practice research network (network of office-based physicians) was considered as a potential mechanism for collecting data on the issues relevant to outpatient psychiatric practice without introducing the biases inherent in tertiary setting-based designs. Such networks have been successfully organized in other medical specialties.

The *Committee on Research Training*, Ronald O. Rieder, M.D., chairperson, focuses on the recruitment and development of psychiatric researchers through a variety of activities. Following the success of their recent workshops at APA annual meetings (a 1991 workshop on overcoming obstacles to starting research and a 1992 workshop on mentorship), members of the committee have proposed the workshop "The Joy of Research" for the 1993 APA annual meeting. In response to the particular need for helping women become psychiatric researchers, the committee submitted the report "Women in Academic Psychiatry and Research" through the Council on Research to the APA Board of Trustees for approval. Members of the committee are in the process of planning a curriculum for psychiatric research and a national conference for research trainees. Committee members provided guidance to APA staff concerning collection of data for the second edition of the *Directory of Research Fellowship Opportunities in Psychiatry*, which was published and distributed by the Office of Research in September 1992. This resource includes listings for 147 research fellowship programs at 70 institutions and five multi-institutional programs in the United States and Canada. To collect data for the directory, during the summer of 1992 APA staff asked chairpersons of departments of psychiatry, directors of psychiatric residency training programs, and research fellowship directors for information about programs in which formal postresidency research in

psychiatry is the principal focus. The directory includes the responses received, as well as material about APA programs and research careers. The introduction to the directory also includes the "Guide for Residents and Fellows Seeking Research Training Opportunities" developed by the Committee on Research Training in September 1991; this guide is designed to help trainees acquire information about and evaluate the opportunities available for research in various departments of psychiatry. APA staff will continue to distribute the *Directory of Research Fellowship Opportunities in Psychiatry* through the Psychiatric Research Training Clearinghouse and to respond to requests for it from students, psychiatric residents, postresidency fellows, and those responsible for their training.

The *Committee on the Biographical Directory and Research on Psychiatric Professional Activities*, Lorrin Koran, M.D., chairperson, believes that APA should continue to collect relevant data from professional activities surveys. The committee plans to present a proposal addressing this need to the council in May 1993.

The committee continues to analyze the data collected from the 1988 professional activities survey and has provided data for the study of specific topics to the Committee on the Chronically Mentally Ill and to the Task Force on Psychiatrist Payment. The committee's article describing basic survey findings was published in the *Journal* in November 1992 (2), and the committee is collaborating with researchers at Harvard University, Johns Hopkins University, and the University of Arkansas on analysis and dissemination to APA and others of additional survey findings on policy issues. In addition, "datagrams" are being developed for publication.

The *Committee on Psychiatric Diagnosis and Assessment*, Layton McCurdy, M.D., chairperson, oversees diagnostic assessment activities, including the work of the Task Force on DSM-IV. As work on DSM-IV nears completion, the committee continues to consider the linkage between ICD-10 and DSM-IV, the forensic implications of DSM-IV, and efforts to ensure gender and cultural sensitivity across the diagnostic classifications. The committee has also recently received information from the APA Division of Government Relations regarding the definition of "serious/severe" mental illness in recent legislation and is considering the implications of these definitions for the treatment of psychiatric illnesses.

The *Task Force on DSM-IV*, Allen Frances, M.D., chairperson, is making final decisions regarding diagnostic criteria for DSM-IV. Data collection from the 12 comprehensive field trial projects, funded by NIMH, has been completed, and analyses to assist the DSM-IV work groups in their final decisions continue to be developed. Results from

the data reanalysis funded by the John D. and Catherine T. MacArthur Foundation have also been circulated to the work groups for their consideration. The results of the field trials, the results of the data reanalyses, and the literature reviews that were completed at the beginning of the revision process will be published in *DSM-IV Sourcebook*. The first volume of five will be available in mid-1993 and will contain the literature reviews from several of the work groups. The task force expects to submit DSM-IV to the Board and Assembly for their approval in the spring of 1993. The task force is also overseeing the work of the DSM-IV Primary Care Work Group, which has begun to produce preliminary drafts for how the disorders will be formatted in the primary care version of DSM-IV. The task force sponsors a bimonthly column on DSM-IV in *Hospital and Community Psychiatry* and a column in *DSM-IV Update*, a semiannual newsletter. Individuals wishing to receive copies of *DSM-IV Update* should write to the APA Office of Research.

The *Task Force on Traumatic Brain Injury*, Stuart Yudofsky, M.D., chairperson, was established in 1990 and concentrates its efforts on the education of physicians. The first effort of the task force was a thorough review of the scientific literature with regard to the epidemiology, diagnosis, treatment, and prevention of psychiatric conditions associated with traumatic brain injury. In the September 1992 issue of *Psychiatric Research Report* the task force summarized its findings from the literature review and from workshops held for practitioners at the past two APA annual meetings. Among the many conclusions reached, the task force found that there has been a far greater emphasis on the assessment and treatment of sensory and motor sequelae of traumatic brain injury than on psychiatric aspects. Furthermore, the psychosocial and cognitive deficits of traumatic brain injury are more commonly a major source of disability to the victims and stress to their families than the sensorimotor concomitants.

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THE AMERICAN JOURNAL OF PSYCHIATRY

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Ethical and legal considerations require careful attention to the protection of a patient's anonymity in case reports and elsewhere. Identifying information such as names, initials, hospital numbers, and dates must be avoided. Also, authors should disguise identifying information when discussing patients' characteristics and personal history.

Informed Consent

Manuscripts that report the results of experimental investigation with human subjects must include a statement that informed consent was obtained after the procedure(s) had been fully explained. In the case of children, authors are asked to include information about whether the child's assent was obtained.

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All papers are reviewed to determine the originality, validity, and importance of content and conclusions. In addition to the regular review process, peer review for statistical content may be required for some manuscripts. This will be determined by the *Journal's* Statistical Editors. Authors will be sent reviewer comments that are judged to be useful to them. All reviewers remain anonymous. Once the Editor has made a final decision on a paper, the authors will be informed.

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Case reports (single or series) should be submitted as Letters to the Editor. All case reports will be peer reviewed. Reports of successfully treated patients must include data on the number of patients treated unsuccessfully by the same method, with an indication of the temporal order of the successes and failures.

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Papers may be submitted before the annual meeting, but they cannot be published until after the meeting. They must be accompanied by a statement that they are in final form. These papers receive the same peer review as other papers and must meet the requirements for one of the types of articles specified in the next section. The *Journal* no longer maintains right of first refusal for annual meeting papers.

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These are usually overview articles that bring together important information on a topic of general interest to psychiatry. Authors who have ideas for such articles are advised to check with the Editorial Office to ensure that a similar work has not already been submitted. Special Articles may not exceed 7,500 words, including an abstract of no more than 250 words, references, tables, and figures (to determine word equivalence, see section on Tables and Figures). This section is not intended to be a forum for the presentation of new data.

Regular Articles

Regular Articles are original communications of scientific excellence in psychiatric medicine and advances in clinical research, containing new data derived from a sizable series of patients. Regular Articles may not exceed 3,800 words, including an abstract of no more than 250 words, references, tables, and figures (to determine word equivalence, see section on Tables and Figures).

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Clinical and Research Reports present 1) data from pilot or uncontrolled studies with suggestive findings warranting further, more definitive investigation, 2) worthwhile replication studies, and 3) clinical studies involving a small number of patients. Essays, program descriptions, literature reviews, and case reports do not meet the criteria for this section. These articles may not exceed 1,300 words, including an abstract of no more than 60 words, references, tables, and figures (to determine word equivalence, see section on Tables and Figures).

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Letters to the Editor. Brief letters (maximum of 500 words, including references; no tables or figures) will be considered

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All parts of the manuscript or letter to the Editor, including case reports, quotations, references, and tables, must be double-spaced throughout. Manuscripts must be typed in upper- and lowercase on one side only of 8.5×11 inch nonerasable bond paper. All four margins must be 1.5 inches. The manuscript should be arranged in the following order, with each item beginning a new page: 1) title page, 2) abstract, 3) text, 4) references, and 5) tables and/or figures. All pages must be numbered. The original and four copies of a manuscript, including tables and figures, should be submitted.

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Title. The title should be informative and as brief as possible. Two-part titles should be avoided.

Byline. Authors listed in the byline should be limited to principal researchers and/or writers; collaborators may be acknowledged in a footnote. Authors' first names are preferred to initials. Degrees should be included after each author's name. The *Journal* subscribes to the Uniform Requirements for Manuscripts Submitted to Biomedical Journals (N Engl J Med 1991; 324:424-428) for authorship summarized here:

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Only those with key responsibility for the material in the article should be listed as authors; others contributing to the work should be recognized separately. Editors may require authors to justify the assignment of authorship.

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Authors of review articles must include the following information, under the headings indicated: **Objective**—the primary purpose of the review article; **Method**—data sources, study selection (the number of studies selected for review and how they were selected), data extraction (rules for abstracting data and how they were applied); **Results**—methods of data synthesis, key findings; and **Conclusions**—including potential applications and research needs. Authors of research articles must include **Objective**—questions addressed by the study; **Method**—design of the study, setting (location and level of clinical care), patients or participants (manner of selection and number who entered and completed the study), interventions (if any), main outcome measures (primary study outcome measure as planned before data collection); **Results**—key findings; and **Conclusions**—including direct clinical applications. Other types of articles, including Clinical and Research Reports, should include unstructured abstracts.

The abstract is a single paragraph no longer than 250 words for Special Articles and Regular Articles and no longer than 60 words for Clinical and Research Reports.

Text

Research design and statistics. The following information regarding research design should be included: 1) a clearly stated hypothesis, 2) the names of the statistical tests used, 3) whether tests were one- or two-tailed, and 4) what test was used for each set of data. Standard deviations, rather than standard errors of the mean, are required. Statistical tests that are not well known should be referenced. All significant and important nonsignificant results must include the test value, degree(s) of freedom, and probability. For example, "The analysis of variance indicated that those who abstained from coffee had significantly higher course grades than those who did not abstain ($F=4.32$, $df=3$, 17 , $p<0.05$).” Reviewers will evaluate the appropriateness of the analyses.

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References are numbered and listed by their order of appearance in text; the text citation is followed by the appropriate reference number in parentheses. Do not arrange the list alphabetically. References in tables and figures are numbered as though the tables and figures were part of the text.

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Type references in the style shown below, **double-spaced throughout**. List all authors; do not use "et al." Abbreviations of journal names should conform to the style used in *Index Medicus*; journals not indexed there should not be abbreviated.

1. Noyes R Jr, DuPont RL Jr, Pecknold JC, Rifkin A, Rubin RT, Swinson RP, Ballenger JC, Burrows GD: Alprazolam in panic disorder and agoraphobia, results from a multicenter trial, II: patient acceptance, side effects, and safety. *Arch Gen Psychiatry* 1988; 45:423-428
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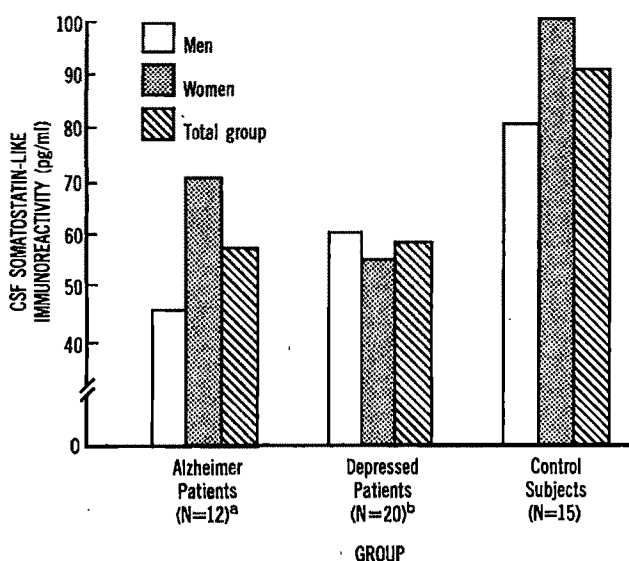
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Tables should be double-spaced, no wider than 120 type-writer characters, including spaces, and no longer than 70 lines. Values expressed in the same unit of measurement should read down, not across; when percentages are given, the appropriate numbers must also be given.

FIGURE 1. Mean CSF Somatostatin Levels of Patients With Alzheimer's Disease, Older Depressed Patients, and Age-Matched Control Subjects



^aSignificant difference between men and women ($t=1.81$, $df=10$, $p<0.05$) and between the total Alzheimer's disease group and the total control group ($t=2.49$, $df=25$, $p<0.01$).

^bSignificant difference between the total depressed group and the total control group ($t=2.75$, $df=33$, $p<0.005$).

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Figures are considered as text and are subject to revision by the authors upon recommendation of the Editors. Figures should, however, be professionally prepared. Glossy or other camera-ready prints should accompany the submitted manuscript. Computer-generated figures that do not meet quality printing standards will be returned for revision. All figure titles and footnotes should be typed and sent together on a separate page.

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Format. Figures are visual expressions of data trends or relationships. Figures that represent numerical data which could be expressed more succinctly or clearly in tabular form should be converted to tables. Line graphs should show change in continuous variables; comparisons of like values in different groups should be presented as bar graphs.

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9. Multiple figures for the same article should be prepared as a set, and the type should be approximately the same size after reduction.

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The Harvard Medical School Department of Psychiatry at McLean Hospital and Massachusetts General Hospital invites applicants to apply for a two year full-time training opportunity in psychiatric neuroscience. The program consists of advanced lectures in the basic and clinical neurosciences, research training under the direction of a faculty mentor and supervised clinical work.

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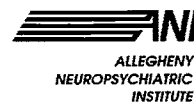
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Allegheny Neuropsychiatric Institute, a 94-bed teaching hospital specializing in psychiatric treatment of patients with concomitant neurological disorders, is seeking an academic clinician to serve as Director of the Traumatic Brain Injury Program. This well-established and fully staffed program provides comprehensive care to patients of all ages with traumatic brain injury, and is comprised of a dedicated 24-bed inpatient unit and outpatient specialty clinics (150 patients). Plans are also under way for development of partial hospitalization and transitional living facilities. Allegheny Neuropsychiatric Institute is a major clinical, teaching and research site within the rapidly expanding Department of Psychiatry at the Medical College of Pennsylvania, Allegheny Campus.

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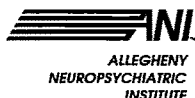
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C. Edward Coffey, M.D., Clinical Director
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THE MANFRED S. GUTTMACHER A W A R D

The American Psychiatric Association and the American Academy of Psychiatry and the Law invite submissions for the 1994 Manfred S. Guttmacher Award. This award is given for an outstanding contribution to the literature of forensic psychiatry in the form of a book, monograph, paper, or any other work presented at a professional meeting or published between May 1, 1992 and April 30, 1993. The award will be formally presented in May 1994 at the American Academy of Psychiatry and the Law meeting held in conjunction with the American Psychiatric Association Annual Meeting in San Francisco, CA. The award includes an honorarium of \$500 and a plaque. The travel expenses of non-member winners will be reimbursed up to \$500. The recipient is expected to present an award lecture. The award will be cited in the Convocation of Fellows program of the 1994 APA Annual Meeting and the lecture will be listed in the APA Convocation Program.

Anyone wishing to apply should submit 6 copies of the work, along with 6 copies of an abstract, to:

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Guttmacher Award Board
American Psychiatric Association
1400 K Street, N.W., Suite 327
Washington, DC 20005

Entries must be received by May 15, 1993. Entries will be acknowledged but not returned.

INDEX TO ADVERTISERS FEBRUARY 1993

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For IM Injection Only

Brief Summary

The following is a brief summary only. Before prescribing, see complete prescribing information in HALDOL and HALDOL Decanoate product labeling.

CONTRAINDICATIONS: Since the pharmacologic and clinical actions of HALDOL Decanoate 50 and HALDOL Decanoate 100 are attributed to HALDOL, haloperidol as the active medication, CONTRAINDICATIONS, WARNINGS, and additional information are those of HALDOL, modified to reflect the prolonged action. HALDOL is contraindicated in severe toxic central nervous system depression or comatose states from any cause and in individuals who are hypersensitive to this drug or have Parkinson's disease.

WARNINGS: Tardive Dyskinesia: Tardive dyskinesia, a syndrome consisting of potentially irreversible, involuntary, dyskinetic movements may develop in patients treated with antipsychotic drugs. Although the prevalence of the syndrome appears to be highest among the elderly, especially elderly women, it is impossible to rely upon prevalence estimates to predict, at the inception of antipsychotic treatment, which patients are likely to develop the syndrome. Whether antipsychotic drug products differ in their potential to cause tardive dyskinesia is unknown. Both the risk of developing tardive dyskinesia and the likelihood that it will become irreversible are believed to increase as the duration of treatment and the total cumulative dose of antipsychotic drugs administered to the patient increase. However, the syndrome can develop, although much less commonly, after relatively brief treatment periods at low doses. There is no known treatment for established cases of tardive dyskinesia, although the syndrome may remit, partially or completely, if antipsychotic treatment is withdrawn. Antipsychotic treatment, itself, however, may suppress (or partially suppress) the signs and symptoms of the syndrome and thereby may possibly mask the underlying process. The effect that symptomatic suppression has upon the long-term course of the syndrome is unknown. Given these considerations, antipsychotic drugs should be prescribed in a manner that is most likely to minimize the occurrence of tardive dyskinesia. Chronic antipsychotic treatment should generally be reserved for patients who suffer from a chronic illness that 1) is known to respond to antipsychotic drugs, and 2) for whom alternative, equally effective, but potentially less harmful treatments are not available or appropriate. In patients who do require chronic treatment, the smallest dose and the shortest duration of treatment producing a satisfactory clinical response should be sought. The need for continued treatment should be reassessed periodically. If signs and symptoms of tardive dyskinesia appear in a patient on antipsychotics, drug discontinuation should be considered. However, some patients may require treatment despite the presence of the syndrome. (For further information about the description of tardive dyskinesia and its clinical detection, please refer to ADVERSE REACTIONS.)

Neuroleptic Malignant Syndrome (NMS): A potentially fatal symptom complex sometimes referred to as Neuroleptic Malignant Syndrome (NMS) has been reported in association with antipsychotic drugs. Clinical manifestations of NMS are hyperpyrexia, muscle rigidity, altered mental status (including catatonic signs) and evidence of autonomic instability (irregular pulse or blood pressure, tachycardia, diaphoresis, and cardiac dysrhythmias). Additional signs may include elevated creatine phosphokinase, myoglobinuria (rhabdomyolysis) and acute renal failure. The diagnostic evaluation of patients with this syndrome is complicated. In arriving at a diagnosis, it is important to identify cases where the clinical presentation includes both serious medical illness (e.g., pneumonia, systemic infection, etc.) and untreated or inadequately treated extrapyramidal signs and symptoms (EPS). Other important considerations in the differential diagnosis include central anticholinergic toxicity, heat stroke, drug fever and primary central nervous system (CNS) pathology. The management of NMS should include 1) immediate discontinuation of antipsychotic drugs and other drugs not essential to concurrent therapy, 2) intensive symptomatic treatment and medical monitoring, and 3) treatment of any concomitant serious medical problems for which specific treatments are available. There is no general agreement about specific pharmacological treatment regimens for uncomplicated NMS. If a patient requires antipsychotic drug treatment after recovery from NMS, the potential reintroduction of drug therapy should be carefully considered. The patient should be carefully monitored, since recurrences of NMS have been reported. Hyperpyrexia and heat stroke, not associated with the above symptom complex, have also been reported with HALDOL.

Usage in Pregnancy: (see PRECAUTIONS - Usage in Pregnancy)

Combined Use With Lithium: (see PRECAUTIONS-Drug Interactions)

General: Bronchopneumonia, sometimes fatal, has followed use of antipsychotic drugs, including haloperidol. Prompt remedial therapy should be instituted if dehydration, hemoconcentration or reduced pulmonary ventilation occur, especially in the elderly. Decreased serum cholesterol and/or cutaneous and ocular changes have been reported with chemically-related drugs, although not with haloperidol. See PRECAUTIONS - Information for Patients for information on mental and/or physical abilities and on concomitant use with other substances.

PRECAUTIONS: Administer cautiously to patients: (1) with severe cardiovascular disorders, due to the possibility of transient hypotension and/or precipitation of anginal pain (if a vasopressor is required, epinephrine should not be used since HALDOL may block its vasopressor activity and paradoxical further lowering of blood pressure may occur; metaraminol, phenylephrine or norepinephrine should be used); (2) receiving anticonvulsant medications, with a history of seizures, or with EEG abnormalities, because HALDOL may lower the convulsive threshold. If indicated, adequate anticonvulsant therapy should be concomitantly maintained; (3) with known allergies or a history of allergic reactions to drugs; (4) receiving anticoagulants, since an isolated instance of interference occurred with the effects of one anticoagulant (phenindione). Concomitant antiparkinson medication, if required, may have to be continued after HALDOL is discontinued because of different excretion rates; if both are discontinued simultaneously, extrapyramidal symptoms may occur. Intraocular pressure may increase when anticholinergic drugs, including antiparkinson drugs, are administered concomitantly with HALDOL. When HALDOL is used for mania in bipolar disorders, there may be a rapid mood swing to depression. Severe neurotoxicity may occur in patients with tyrotoxicosis receiving antipsychotic medication, including HALDOL.

The 1, 5, 10 mg HALDOL tablets contain FD&C Yellow No. 5 (tartrazine) which may cause allergic-type reactions (including bronchial asthma) in certain susceptible individuals, especially in those who have aspirin hypersensitivity.

Information for Patients: Mental and/or physical abilities required for hazardous tasks or driving may be impaired. Alcohol should be avoided due to possible additive effects and hypotension.

Drug Interactions: Patients receiving lithium plus haloperidol should be monitored closely for early evidence of neurological toxicity and treatment discontinued promptly if such signs appear. As with other antipsychotic agents, it should be noted that HALDOL may be capable of potentiating CNS depressants such as anesthetics, opiates, and alcohol.

Carcinogenesis, Mutagenesis and Impairment of Fertility: No mutagenic potential of haloperidol was found in the Ames Salmonella microsome activation assay. Negative or inconsistent positive findings have been obtained in *in vitro* and *in vivo* studies of effects of haloperidol on chromosome structure and number. The available cytogenetic evidence is considered too inconsistent to be conclusive at this time. Carcinogenicity studies using oral haloperidol were conducted in Wistar rats (dosed at up to 5 mg/kg daily for 24 months) and in Albino Swiss mice (dosed at up to 5 mg/kg daily

for 18 months). In the rat study survival was less than optimal in all dose groups, reducing the number of rats at risk for developing tumors. However, although a relatively greater number of rats survived to the end of the study in high dose male and female groups, these animals did not have a greater incidence of tumors than control animals. Therefore, although not optimal, this study does suggest the absence of a haloperidol related increase in the incidence of neoplasia in rats at doses up to 20 times the usual daily human dose for chronic or resistant patients. In female mice at 5 and 20 times the highest initial daily dose for chronic or resistant patients, there was a statistically significant increase in mammary gland neoplasia and total tumor incidence; at 20 times the same daily dose there was a statistically significant increase in pituitary gland neoplasia. In male mice, no statistically significant differences in incidences of total tumors or specific tumor types were noted. Antipsychotic drugs elevate prolactin levels; the elevation persists during chronic administration. Tissue culture experiments indicate that approximately one-third of human breast cancers are prolactin dependent *in vitro*, a factor of potential importance if the prescription of these drugs is contemplated in a patient with a previously detected breast cancer. Although disturbances such as galactorrhea, amenorrhea, gynecostasia, and impotence have been reported, the clinical significance of elevated serum prolactin levels is unknown for most patients. An increase in mammary neoplasms has been found in rodents after chronic administration of antipsychotic drugs. Neither clinical studies nor epidemiologic studies conducted to date, however, have shown an association between chronic administration of these drugs and mammary tumorigenesis; the available evidence is considered too limited to be conclusive at this time.

Usage in Pregnancy: Pregnancy Category C. Safe use in pregnancy or in women likely to become pregnant has not been established; use only if benefit clearly justifies potential hazards to the fetus.

Nursing Mothers: Infants should not be nursed during drug treatment.

Pediatric Use: Controlled trials to establish the safety and effectiveness of intramuscular administration in children have not been conducted.

ADVERSE REACTIONS: Adverse reactions following the administration of HALDOL Decanoate 50 or HALDOL Decanoate 100 are those of HALDOL haloperidol. Since vast experience has accumulated with HALDOL, the adverse reactions are reported for that compound as well as for haloperidol decanoate. As with all injectable medications, local tissue reactions have been reported with haloperidol decanoate.

CNS Effects: Extrapyramidal Symptoms (EPS) — EPS during the administration of HALDOL (haloperidol) have been reported frequently, often during the first few days of treatment. EPS can be categorized generally as Parkinson-like symptoms, akathisia, or dystonia (including opisthotonos and oculogyric crisis). While all can occur at relatively low doses, they occur more frequently and with greater severity at higher doses. The symptoms may be controlled with dose reductions or administration of antiparkinson drugs such as benztropine mesylate USP or trihexyphenidyl hydrochloride USP. It should be noted that persistent EPS have been reported; the drug may have to be discontinued in such cases. **Withdrawal Emergent Neurological Signs** — Abrupt discontinuation of short-term anti-psychotic therapy is generally uneventful. However, some patients on maintenance treatment experience transient dyskinetic signs after abrupt withdrawal. In certain cases these are indistinguishable from "Tardive Dyskinesia" except for duration. It is unknown whether gradual withdrawal will reduce the occurrence of these signs, but until further evidence is available HALDOL should be gradually withdrawn. **Tardive Dyskinesia** — As with all antipsychotic agents HALDOL has been associated with persistent dyskinesias. Tardive dyskinesia, a syndrome consisting of potentially irreversible, involuntary, dyskinetic movements, may appear in some patients on long-term therapy or may occur after drug therapy has been discontinued. The risk appears to be greater in elderly patients on high-dose therapy, especially females. The symptoms are persistent and in some patients appear irreversible. The syndrome is characterized by rhythmic involuntary movements of tongue, face, mouth or jaw (e.g., protrusion of tongue, puffing of cheeks, puckering of mouth, chewing movements). Sometimes these may be accompanied by involuntary movements of extremities and the trunk. There is no known effective treatment for tardive dyskinesia; antiparkinson agents usually do not alleviate the symptoms of this syndrome. It is suggested that all antipsychotic agents be discontinued if these symptoms appear. Should it be necessary to reinstitute treatment, or increase the dosage of the agent, or switch to a different antipsychotic agent, this syndrome may be masked. It has been reported that fine vermicular movement of the tongue may be an early sign of tardive dyskinesia and if the medication is stopped at that time the full syndrome may not develop. **Tardive Dystonia** — Tardive dystonia, not associated with the above syndrome, has also been reported. Tardive dystonia is characterized by delayed onset of choreic or dystonic movements, is often persistent, and has the potential of becoming irreversible. **Other CNS Effects** — Insomnia, restlessness, anxiety, euphoria, agitation, drowsiness, depression, lethargy, headache, confusion, vertigo, grand mal seizures, and exacerbation of psychotic symptoms including hallucinations, and catatonic-like behavioral states which may be responsive to drug withdrawal and/or treatment with anticholinergic drugs.

Body as a Whole: Neuroleptic malignant syndrome (NMS), hyperpyrexia and heat stroke have been reported with HALDOL. (See WARNINGS for further information concerning NMS.)

Cardiovascular Effects: Tachycardia, hypotension, hypertension and ECG changes, including prolongation of the Q-T interval and ECG pattern changes compatible with the polymorphous configuration of torsades de pointes.

Hematologic Effects: Reports of mild, usually transient leukopenia and leukocytosis, minimal decreases in red blood cell counts, anemia, or a tendency toward lymphomonocytosis; agranulocytosis rarely reported and only in association with other medication.

Liver Effects: Impaired liver function and/or jaundice.

Dermatologic Reactions: Maculopapular and acneiform reactions, isolated cases of photosensitivity, loss of hair.

Endocrine Disorders: Lactation, breast engorgement, mastalgia, menstrual irregularities, gynecostasia, impotence, increased libido, hyperglycemia, hypoglycemia and hyponatremia.

Gastrointestinal Effects: Anorexia, constipation, diarrhea, hyper-salivation, dyspepsia, nausea and vomiting.

Autonomic Reactions: Dry mouth, blurred vision, urinary retention, diaphoresis, and priapism.

Respiratory Effects: Laryngospasm, bronchospasm and increased depth of respiration.

Special Senses: Cataracts, retinopathy and visual disturbances.

Other: Cases of sudden and unexpected death have been reported in association with the administration of HALDOL. The nature of the evidence makes it impossible to determine definitively what role, if any, HALDOL played in the outcome of the reported cases. The possibility that HALDOL caused death cannot, of course, be excluded, but it is to be kept in mind that sudden and unexpected death may occur in psychotic patients when they go untreated or when they are treated with other antipsychotic drugs.

Postmarketing Events: Hyperammonemia has been reported in a 5½ year old child with citrullinemia, an inherited disorder of ammonia excretion, following treatment with HALDOL.

IMPORTANT: Full directions for use should be read before HALDOL or HALDOL Decanoate products are administered or prescribed.

For information on symptoms and treatment of overdosage, see full prescribing information. The short-acting HALDOL injectable form is intended only for acutely agitated psychotic patients with moderately severe to very severe symptoms.

12/30/91

Reference:

1. Youssef HA. A five-year follow-up study of chronic schizophrenics and other psychotics treated in the community: depot haloperidol decanoate versus other neuroleptics. *Adv Ther.* 1989;6(4):186-195.

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During dose adjustment or episodes of exacerbation of psychotic symptoms, therapy with HALDOL Decanoate 100 or HALDOL Decanoate 50 can be supplemented with short-acting forms of HALDOL[®] (haloperidol). The side effects of the decanoate products are those of HALDOL. The prolonged action of HALDOL Decanoate 100 and HALDOL Decanoate 50 should be considered in the management of side effects.

Please see brief summary of Prescribing Information on adjacent page.